Dissertation on

"A STUDY OF SERUM URIC ACID AT PRESENTATION AS AN INDICATOR OF OUTCOME AMONG ACUTE ISCHEMIC STROKE PATIENTS"

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CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY OF SERUM URIC ACID AT PRESENTATION AS AN INDICATOR OF OUTCOME AMONG ACUTE ISCHEMIC STROKE PATIENTS"** is a bonafide original work done by **Dr.SANKARA AVUDAYAPPAN A.P** in partial fulfillment of the requirements for M.D.GENERAL MEDICINE BRANCH – I examination of the Tamilnadu Dr. M.G.R Medical University to be held in April 2017, under my guidance and supervision in 2016.

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ABSTRACT

Background and objectives

Among all the neurological diseases of adult life, the cerebrovascular ones clearly rank the first in frequency and importance. At least 50% of the neurological disorders in a general hospital are of this type. Stroke, after heart disease and cancer is one of the most common causes of death.

Uric acid is the most abundant aqueous antioxidant in humans, and contributes as much as two-thirds of all free radical scavenging capacity in plasma. A number of major epidemiological studies have identified high UA concentrations as an important risk marker for stroke in unselected populations. There is a pressing need to identify additional treatable risk factors for stroke that are easily measured and highly prevalent in the general population. Hyperuricemia is one such potential risk factor. This study aims at finding the relationship between serum uric acid levels at presentation and the clinical outcome following acute ischemic stroke so that appropriate intervention can be targeted to reduce the risk and improve the outcome following acute stroke.

Methods

Patients admitted in general wards of Department of Medicine, Madras Medical College, Chennai, with acute ischemic stroke during the period of

April 1st 2016 to September 31st 2016 were taken up for study considering the inclusion and exclusion criteria.

Results

Majority of this stroke population were between 61 to 70yrs old. Hypertension constituted the major risk factor in this stroke population followed by Diabetes mellitus, smoking, CAD, and dyslipidemia for hypertension and male sex. Overall 63% 0f patients had bad outcome (39 out of 60). Among them 92% (34 out of 39) had high serum uric acid levels. When serum uric acid levels were correlated with outcome at 7 days it was found to be associated with poor outcome.

Interpretation and conclusion

Hypertension, Diabetes mellitus, smoking, CAD, and dyslipidemia were the most important risk factors associated with stroke. Hypertension and male sex were significantly associated with serum uric acid levels in stroke patients. Elevated uric acid level was associated with poor outcome among stroke patients at 7 days follow up period.

Key words; cerebrovascular accident, serum uric acid, anti-oxidants, ischemia, prognosis Hypertension, outcome

LIST OF ABBREVIATIONS

CVA	:	CerebroVascular Accident
TIA	:	Transient Ischemic Attack
WHO	:	World health organisation
CBF	:	Cerebral blood flow
CMRO	:	Cerebral Metabolic rate for oxygen
CVR	:	Cerebral Vascular resistance
ICP	:	Intracranial pressure
ECG	:	Electrocardiography
ATP	:	Adenosine triphosphate
OEF	:	Oxygen extraction fraction
PAF	:	Platelet Activating Factor
BP	:	Blood pressure
HGPRT	:	Hypoxanthine guanine phosphoribosyltransferase
PRPP	:	Phospho-ribosyl pyrophosphate
XDH	:	Xanthine Dehydrogenase
XO	:	Xanthine Oxidase
UA	:	Uric acid
SUA	:	Serum uric acid
NO	:	Nitric oxide

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INTRODUCTION

INTRODUCTION

Uric acid is found to be an abundant anti oxidant in plasma having its role in eliminating hydroxyl and superoxide radicals in the tissues. It is also proved to be having pro oxidant properties in the absence of other related anti oxidants.Hence a detailed study regarding its role is necessary in the etiology of various vascular events.

Uric acid levels and its role in endothelial dysfunction, dyslipidemia, diabetes, CAD, Stroke and Metabolic syndrome are under study around the world. Our study is based on its association with outcome in ischemic stroke patients. Stroke ranks next to Heart disease and Cancer as the leading cause of death. Hence its of prime importance in fixing its role in vascular events.

In ischemic stroke patients serum uric acid levels were assessed at the time of presentation and patients are followed up for next seven days for assessing their outcome based on GLASGOW OUTCOME SCALE.

Previous larger data involving NHANES study clearly proved an independent association between high uric acid levels and poor outcome.

Hence serum uric acid measurements may play a major role in treatment aspects of patients with ischemic stroke in the future..

AIMS AND OBJECTIVES

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To study the association between serum uric acid at presentation and the clinical outcome among acute ischemic stroke patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Cerebro vascular diseases includes ischemic stroke and hemorrhagic stroke accounting for second leading cause of death world wide. A stroke or cerebro vascular accident is an abrupt onset of a neurological deficit due to a focal vascular cause[1] The definition of stroke goes by clinical and laboratory studies including brain imaging to support the diagnosis. Generally cerebral ischemia is due to reduction in cerebral blood flow that lasts longer thahn several seconds, but symptoms are manifested within seconds since neurons lack glycogen. If blood flow gets restored quickly symptoms are only transient; this is said to be Transient Ischemic Attack(TIA). Hence the definition of TIA is neurological symptoms and signs that resolve within 24 hours without any evidence of brain infarction on imaging. If Signs & symptoms lasts more than 24 hours or brain infarction is demonstrated it is said to be a stroke.

Classification of CerebroVascular Diseases by WHO-

Cerebral Thrombosis / Embolism – 85%

1 Cerebral Hemorrhage – 10%

2 Sub Arachnoid Hemorrhage – 2%

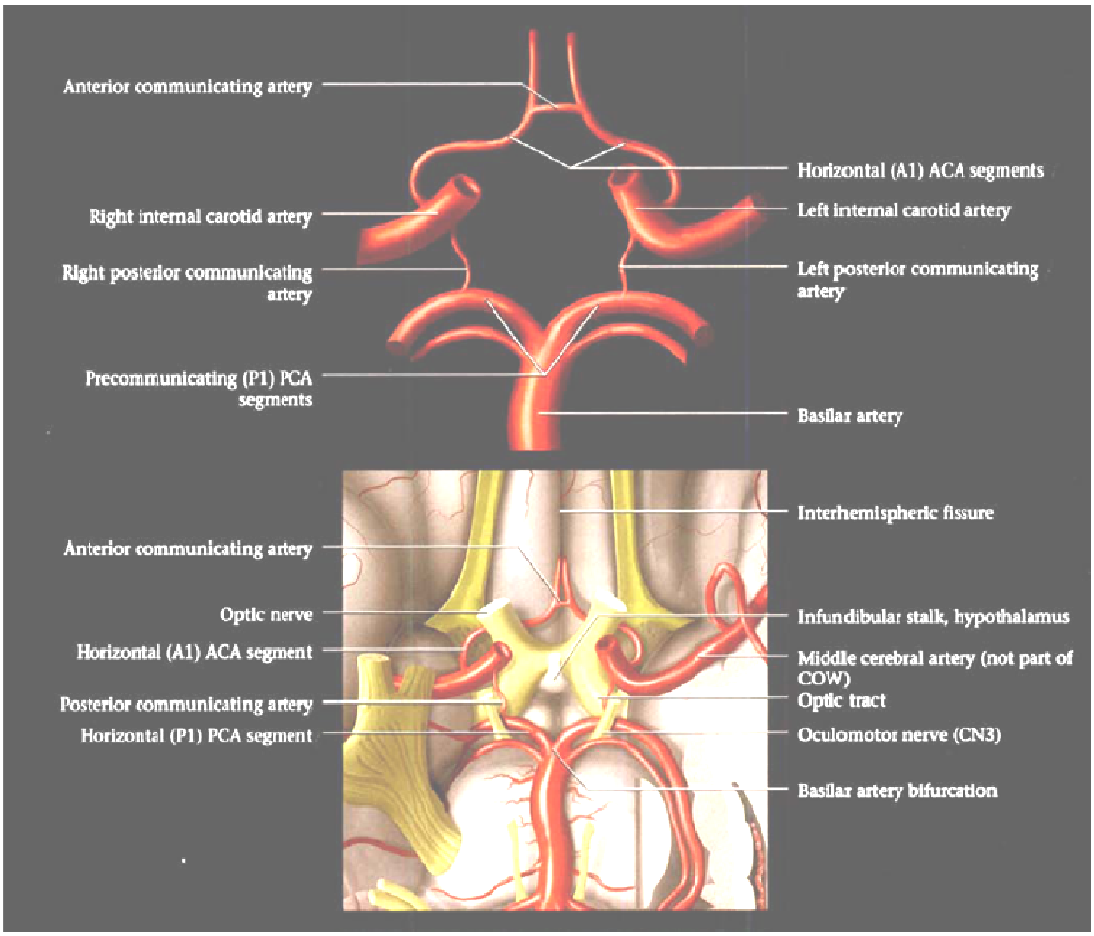
Hence Ischemic stroke contributes to the most frequent form of stroke. It is divided into Thrombotic and Embolic strokes; Thrombotic – 25%; Embolic – 75% of all ischemic strokes respectively.

ANATOMY AND PHYSIOLOGY OF CEREBRAL CIRCULATIONS

Brain tissues need continuous adequate blood supply which on its cessation for a prolonged time leads to irreparable damage to the brain cells.

CEREBRAL VESSELS & NORMAL CEREBRAL BLOOD FLOW

Brain receives blood from the Basilar artery and Internal Carotid artery. Branches of these arteries form CIRCLE OF WILLIS. Venus drainage is by sinuses which open into Interal Jugular vein. Brain receives 750ml to 800ml of blood/ minute. It is about 15% to 16% of total cardiac output and about 50 to 55ml/100gm of brain tissue per minute. The cerebral blood flowwhich is higher in the first decade of life falls at puberty to adult values. Cerebral blood flow decreases from middle age to elderly. Human brain contributes to 2% of the total body weight but it receives 15% of cardiac output. This high flow rate indicates brain's high metabolic rate. It consumes about 3.5ml of Oxygen/ 100gm of Brain tissue/minute.



CIRCLE OF WILLIS

Many anomalies may occur, but Hutchinson and Acheron (1975) concluded that all such abnormalities are of theoretical interest only unless occlusive vascular disease develops, during which collateral circulation may be affected.

The cerebral artery gives rise to two types of branches, cortical and central. Cortical branches are distributed to the cerebral cortex on its surfaces. The central branches enter the substances of the cerebrum through the anterior and posterior matter buried in it. The central branches are end arteries[3].

Blood Supply of Relevant Parts of Brain:

1. Cerebral Cortex

a) In general, by the cortical branches of the three cerebral arteries.

b) Motor Area: by the frontal cortical branches of middle cerebral artery except for the leg area in the upper part of the pre-central gyrus and paracentral lobule and the paracentral cortical branches of the anterior cerebral artery (leg area).

c) Auditory Area: By the cortical branch of the middle cerebral artery.

d) Visual Area: By the occipital branch of the posterior cerebral artery.

e) Speech Area: Both motor and sensory, by the cortical branches of the middle cerebral artery.

2. Midbrain: the branches from the posterior cerebral arteries supply Midbrain, including their central branches, both postero-medial and postero-lateral.

3. Pons: By the pontine branches of the basilar arteries.

4. Medulla: By the Medullary branches of the vertebral artery and branches from the posterior inferior cerebellar artery.

5. Cerebellum: By the superior cerebellar artery is a branch of basilar artery and the posterior inferior cerebellar artery a branch of vertebral artery.

6. Internal Capsule: It is supplied by the central branches of:

a. Middle cerebral artery, the lenticulo striate branches;

b. Anterior cerebral artery, the Heubner's recurrent branch;

c. Posterior communicating artery

d. Anterior choroidal artery

7. Corpus Striatum:

a. It is supplied chiefly by the anterolateral central branches of the middle cerebral artery

b. Partly by the antero-medial branches from the anterior cerebral and anterior communicating arteries.

8. Thalamus:

a. Is supplied chiefly by the postero-medial and postero-lateral central branches of the posterior cerebral artery and

b. Partly by the antero-medial central branches.

Anatomical Peculiarities of Cerebral Arteries:

a. The free anastomoses of the circle of Willis equalize pressure in the arteries of the two sides.

b. The presence of the blood brain barrier permits a selective passage of the blood contents to the nervous tissue and thus the toxic and harmful substances are ordinarily prevented from reaching the brain.

c. Central branches of the cerebral arteries are end arteries. Thrombosis of any one of them therefore will cause infarction. The cortical branches establishing poor anastomoses with each other cannot compensate for any loss of blood supply to a particular area of the cortex.[3,4]

Parameter	Value
CBF(cerebral blood flow)	
Global	45-55 ml/100 gm/min
Cortical (mostly grey matter)	75-80 ml/100 gm/min
Sub cortical (mostly white matter)	Approx. 20 mL/100 gm/min
CMRO (Cerebral Metabolic rate for oxygen)	3-3.5 mL/100 gm/min
CVR (Cerebral Vascular resistance)	1.5-2.1mm Hg/100 gm/min/ml
Cerebral Venous PO2	35-40 mm of Hg
ICP (intracranial pressure)	8-12 mm of Hg

REGULATION OF CEREBRAL BLOOD FLOW

Regulation is by;

1 Autoregulation

2 Chemical factors

3 Neural factors

AUTOREGULATION

Brain regulates its own blood flow by means of autoregulation. It depends on effective perfusion pressure and Cerebral Vascular Resistance. Cerebral blood flow depends on the balance between effective perfusion pressure and the vascular resistance in brain.

EFFECTIVE PERFUSION PRESSURE

It is the balance between mean arterial pressure and venus pressure across the organ divided by resistance. As venus pressure is zero in brain mean arterial pressure plays an important role in regulating cerebral blood flow. Auto regulation is possible if mean arterial pressure is within the range of 60 – 140mm Hg. Auto regulation seems to be due to Intrinsic feature of Cerebral smooth muscles (myogenic)

CEREBRAL VASCULAR RESISTANCE

The blood flow to brain decreases if resistance is more. Resistance to blood flow is by Intracranial pressure, CSF pressure & viscosity of blood. Increase in the Intracranial pressure or CSF pressure tend to decrease cerebral blood flow. These pressures are elevated in Head injury. But severe Ischemic effects due to head injury are avoided by protective reflexes such as Cushing reflex. Cushing reflex is a protective reflex that protects the brain tissue during periods of decreased cerebral blood flow. Compression of cerebral blood vessels decreases the blood flow to vasomotor centre resulting in local hypoxia

and hypercapnea leading to activation of the centre resulting in peripheral vasoconstriction and rise in arterial blood pressure. This helps to restore the cerebral blood flow.

MUNRO KELLIE DOCTRINE

This states that though the cerebral arteries are compressed by increased intracranial pressure or CSF pressure, the volume of brain tissue is not affected, since brain tissue is non-compressible.

VISCOSITY

Viscosity of blood influences cerebral blood flow by increase in viscosity leading to increased cerebro vascular resistance and decreased blood flow. Eg: Polycythemia. Hence in anaemia cerebro vascular resistance is reduced both due to decreased viscosity and reduced oxygen carrying capacity of blood. Thus adequate oxygen delivery in presence of cerebral Ischemia is provided by a hematocrit of 30-35%

CHEMICAL REGULATION OF CEREBRAL BLOOD FLOW

Chemical factors that regulate the blood flow are PaO2, PaCo2& H+ concentration. CO2 is the most important factor as it causes dilation of cerebral blood vessels leading to increased cerebral blood flow. For each 1mm Hg change in PaCO2 changes CBF by 1-2ml/100gm of brain tissue/minute. This response decreases if PaCO2 is below 25mm Hg. As CO2 diffuses rapidly through the cerebro vascular endothelium changes in CBF caused due to

PaCO2 also occurs rapidly. But a moderate increase in PaCO2 does not alter the blood flow due to autoregulation.

PaO2

Hypoxia increases cerebral blood flow by vaso dilatation. CBF increases when PaO2 falls less than 60mm Hg. PaO2 in the range of 60-300 Hg has minimal influence on CBF. High PaO2 values decreases CBF minimally.

NEUROGENIC REGULATION OF CEREBRAL BLOOD FLOW

Neurological influence tend to be high on larger cerebral arteries. Innervations declines directly with the vessel size. Cerebral blood vessels are supplied by sympathetic vaso constrictive fibres. But these fibres do not play any role in regulating CBF under normal conditions. In pathological conditions like hypertension these sympathetic nerves cause constriction of blood vessels leading to reduction in blood flow. Thus it prevents cerebro vascular hemorrhage and cerebral stroke.

CEREBROVASCULAR RESISTANCE(CVR)

It depends on

1. Pressure within the cranium
2. Viscosity of blood
3. Patency of cerebral blood vessels
4. PaCO2
5. PaO2

6. Adrenaline & Nor-Adrenaline
7. Ethanol

PHYSIOLOGY OF CEREBRAL ISCHEMIA

Cerebral infarction consists of

1. Decreased oxygen and glucose supply
2. Alteration of cellular metabolism

The oxygen stores in brain are very minimal but it can derive its energy only from oxidative metabolism of glucose. Hence when CBF decreases various functional and neurophysiological changes dependant on glucose metabolism occurs at various thresholds of flow. Cerebral metabolism is measured by PET scan based on cerebral metabolic rate of glucose and oxygen. Oxygen extraction fraction (OEF)v is the same throughout the brain tissue. In normal human brain CMRO2 reflex cerebral blood flow. During Ischemia OEF increases and CMRO2 falls. These changes are seen early after acute ischemic stroke.

When the flow is restored quickly functional recovery is possible. As of now lactate accumulates due to anaerobic metabolism of glucose leading to fall in ATP synthesis and pH. Cellular transport and neuro transmission also fails. Release of platelet activating factor occurs which is also neuro toxic.

If flow falls further infarction occurs and function does not recover. By this stage CMRO2 and CBF are low and OEF is normal which indicates

decreased metabolic activity. Duration of Ischemia and depth of Ischemia determines the consequences of fall in CBF. Due to collaterals flow is never zero in localised ischemia. CBF is also influenced by cerebral oedema and raised ICP. Vaso dilators are acid metabolites and increased extracellular potassium concentrations. Vasoconstrictors are prostaglandins released from platelets and cell membranes. Metabolic effects of ischemia are increased by high glucose concentration. Seizures may occur due to raised lactate levels. Ischemic brain also demonstrates decreased responses to changes in PaCO2 & PaO2 and impaired autoregulation.

RISK FACTORS FOR STROKE

Major

1. Age
2. Hypertension
3. Sex
4. Family history
5. Cardiac Co-morbidities
6. History of TIA
7. History of smoking
8. Fibrinogen level
9. Hematocrit level
10. Abnormal hemoglobins
11. Cocaine abuse
12. Diabetes

Others

1. Oral Contraceptives use
2. Lifestyle pattern
3. Obesity
4. Hyperlipidemia
5. Hyperuricemia
6. Infections
7. Increased homocysteine levels
8. Race
9. Alcoholism
10. Peripheral vascular disease

UNMODIFIABLE RISK FACTORS

1. Age of the individual
2. Sex
3. Race
4. History of previous stroke

MODIFIABLE RISK FACTORS

1. **Hypertension**

Hypertension is the second most powerful risk factor for Ischemic stroke after age both SBP & DBP are important. Fall in Diastolic BP by 6mm Hg decreases incidence of stroke by 40%.

2. **Myocardial Infarction**

Transneural Infarcts poses higher risk for stroke than sub endocardial infarcts. Previous history of MI is also considered to be a risk factoe for stroke. Risk of stroke doubles in presence of cardiac disease. Cardiac failure, coronary Heart Disease and Angina increases the risk of stroke.

3. Diabetes Mellitus

Impaired glucose tolerance and elevated HbA1C are found to be in 40-50% of patients with cerebral infarct who were not known diabetics.

4 .TIA

Previous history of TIA increases the risk of stroke.

5.SMOKING

Studies have proved that risk of stroke doubles in smokers. Smoking cessation reduces the risk of stroke within next two years.

6.Alcohol

Effects of alcohol depends on its pattern of intake. Chronic minimal intake decreases stroke incidence whereas acute alcohol binge drinking leads to increased risk of cerebral infarction.

7.Lipids

Studies have proved a weak association between total cholesterol and incidence of atherothrombotic brain infarct.

8.Exercise

Risk of stroke increases in women with sedentary lifestyle.

OUTCOME OF STROKE

Demographic factors

Age

Secondary complications including bed sores and pneumonia are increased in elderly with stroke. Hence age has a negative influence on outcome of stroke.

Gender

Males with stroke have a poor outcome compared to females since endogenous estrogens are found to be neuroprotective.

RISK FACTORS

Previous history of stroke and atrial fibrillation are associated with poor outcome.

CLINICAL FINDINGS

Consciousness and Gaze deviation

Presence of gaze deviation and decreased consciousness are associated with poor outcome

Blood pressure

Both hyper and hypotension are deleterious to brain prenchyma. Hypotension decreases flow whereas hypertension can cause hemorrhagic transformation.

Temperature

Rise in temperature increases the levels of excitotoxic neurotransmitters Hence 1* C rise of temperature is associated with two fold increase in relative risk for poor outcome.

COURSE OF THE ILLNESS AND PROGNOSIS

The course of cerebral thrombosis is unpredictable that it may progress and worsen the clinical status as time progresses. Progression is mostly due to extension of mural thrombus. The thrombus may extend and occlude the anastomotic branches. Basilar artery thrombus may extend along its entire length and Carotid thrombus may extend into anterior cerebral artery and prevent flow from opposite side.

Embolic particles from occluded site may propagate and precipitate an abrupt change. Large infarcts may compress the adjacent structures leading to tentorial herniation after days. Inferior cerebellar infarcts may cause fatal foramen magnum herniation.40% mortality is seen in large basilar infarct with deep coma.it is 3% to 5% with smaller infarcts.

If coma or stupor develops further focus of care has to be on preventing bed sores, Aspiration, Controlling Brain edema, Patency of airway, maintaining fluid and electrolyte balance.

Stroke evolves characteristically with flaccid muscles for initial few days. Then gradually spasticity develops and tendon reflexes become brisk. Initially the patient may assume a posture with flexed adducted upper limbs and extended lower limbs. With slow evolution of spasticity the functional recovery may be delayed and may not be complete.

Whereas early development of spasticity or early grasp reflex favours a quick and better outcome. Patients may remain hemiplegic in extensive temporoparietal lesions. Stroke involving thalamus and lentiform nucleus incompletely are associated with hemitremors and hemiataxia and hemi choreoathetosis. Physiotherapy plays a major role in avoiding contractures.

Recurrent epileptic seizures are more common in embolic cortical infarcts compared to thrombotic strokes. Left frontal lobe infarcts are associated with fatigability and reactive depression.

Patients developing thrombotic strokes are prone to develop future similar episodes particularly if hypertensive and diabetic.Multiple infarcts over years may lead to multi infarct dementia.

TREATMENT ASPECTS

Medical treatment available are to decrease the area of infarct and to avoid the adverse effects of increased ICP. Thrombolysis are used in higher centres and anti platelets are used as additives later. Mannitol and oral glycerol are used to reduce the cerebral edema. Other supportive treatments are to reduce the incidence of secondary complication

URIC ACID

Uric acid is a heterocyclic compound of carbon, nitrogen, oxygen and hydrogen with the formula C5H4N4O3. Uric acid is a product of metabolic breakdown of purine nucleotides.

Pathophysiological effects of uric acid keeps on evolving as its role in gout in older days to role in metabolic syndromes recently.The major source of uric acid is its endogenous production .one third are from dietary purines. Uric acid is less soluble and 70% is excreted via kidneys.

PATHOPHYSIOLOGY

Hyperuricemia occurs due to either overproduction or decreased excretion or both. Decreased excretion contributes to most cases.

URIC ACID - URATE + H+

Most uric acid in circulation are in the form of urate anion. Most mammalian species have low levels of uric acid since it has been converted to allantoin by uricase enzyme. Humans lack the gene expression for uricase enzyme leading to levels around 7 mg/dl. Normal adult males have twice the levels of female urate levels. Normally daily turnover of 60% of the urate pool occurs in humans.

Uric acid in the blood is saturated at 6.5 to 6.9 mg/dl. Upper limit of solubility is around 7mg/dl. It is freely filtered at glomerulus and gets reabsorbed in proximal tubules. It also gets secreted in proximal tubules. uric acid secretion in tubules parallels with the serum levels. Thus higher serum levels are associated with marked increase in excretion.

Decreased excretion may be due to decreased filtration at glomerulus or secretion at proximal tubules or increased reabsorption at tubules. Decreased excretion contributes to hyperuricemia of kidney disease. Acidotic conditions like DKA, SALICYLATE OVERDOSE, STARVATION KETOSIS, ALCOHOL IC EXCESS leads to decreased tubular secretion of uric acid. These organic acids compete with uric acid for its secretion. Patients on diuretic therapy or with Diabetes insipidus exhibit enhanced reabsorption of uric acid at site distal to its secretion.

Hyperuricemia due to overproduction may be due to dietary excess of purines or increased breakdown of purine nucleotides. Small proportion of overproducers have enzymatic defects that account for their hyperuricemia. They include Hypoxanthine guanine phosphoribosyl transferase deficiency as in leisch nyhan syndrome which exhibits complete deficiency of the enzyme whereas partial deficiency leads to Kelly Seegmiller sydrome. Increased purine breakdown is seen in rhabdomyolysis, cytotoxic therapy, blast crisis of leukemias. Alcohol consumption contributes to both overproduction and decreased excretion by increased breakdown of ATP and decreased excretion due to organic acids that compete with uric acid for excretion.

Other causes include Glycogenoses type I and Aldolase – B deficiency that also cause increased production and decreased excretion.

URIC ACID AS AN ANTI OXIDANT

Uric acid plays a role as an anti oxidant but is also a marker of oxidative stress.like ascobate it also acts as a peroxidant.It is proposed that hyperuricemia induced oxidative stress plays a roll in etiology of stroke ,atherosclerosis and metabolic syndrome. Ischemia followed by recirculation results in freeradical production. Its production can also occur at low tissue oxygen tension due to accumulation of reduced compounds.

Xanthine Oxidase is the source of free radicals in reperfusion injury .Ischemia promotes the conversion of Xanthine dehydrogenase to Xanthine oxidase. This conversion is also enhanced by increased calcium levels. Nitric

oxide also acts as a source of free radical.rising calcium levels in tissues stimulates nitric oxide synthetase.

Uric acid once considered to be inert is now associated with Gout and Xanthinuria. It acts as an antioxidant by reacting with hydroxyl radicals and hypochlorous acid. Uric acid also preserves the vasodilation by endothelium in situations of oxidative stress.

SERUM URIC ACID IN STROKE

Hypertension And Uric Acid

Uric acid plays a major role in both in pathophysiology of Hypertension.

1. 25% of newly detected hypertensives have an elevated serum uric acid levels.
2. Hypertension leads to decreased renal blood flow which in turn leads to increased urate absorption.
3. Hyperuricemia on chronic basis leads to renal injury leading to hypertension.
4. Uric acid stimulates macrophage infiltration of atherosclerotic vessels.
5. Uric acid in raised levels also leads to inhibition of nitric oxide.

FREE RADICAL INJURY IN CEREBRAL ISCHEMIA

Free radical mediated injury plays a major role in pathogenesis of infarct. Free radicals are liberated from dysfunctional mitochondria, inflammatory cells and excitotoxic mechanisms due to increased glutamate and aspartate levels.

Superoxide and hydroxyl radicals play a major role in injury by causing lipid peroxidation that damages mitochondrial and cell membranes. Lipid peroxidation disrupts enzymes ,receptors and various transporters. Thus local oxidants increase infarct size and injury.

As uric acid plays a role as an anti oxidant it is expected that elevated levels are beneficial but studies have proved that high uric acid levels are deleterious and are not beneficial. Reason is uric acid tend to act as a pro oxidant in the absence of other anti oxidants like ascorbate. Thus reduced ascorbate levels converts uric acid into a pro oxidant rather than as an anti oxidant.

Consistent with this hypothesis it is observed that high uric acid and low ascorbate levels are associated with a poorer outcome.

ENDOTHELIAL DYSFUNCTION

Uric acid promotes LDL-C oxidation in vitro and also stimulates granulocyte adherence to endothelium. Raised uric acid levels are associated with raised inflammatory markers. Uric acid crystallise and gets accumulated within atherosclerotic plaques.

METABOLIC SYNDROME

Serum uric acid is also associated with increased incidence of metabolic syndrome. Both hyperuricemia and metabolic syndrome are related to underlying insulin resistance.

Metabolic syndrome is associated with insulin resistance and reduced insulin induced uricacid excretion leading to hyperuricemia. Insulin resistance is linked to increased purine synthesis and thus leading to hyperuricemia.

Serum uric acid also plays a role in endothelial dysfunction and insulin resistance.Uric acid decreases the bioavailability of nitric oxide but nitric oxide is needed for glucose uptake.Thus hyperuricemia leads to insulin resistance.

URIC ACID AND RENAL DYSFUNCTION

Apart from causing Metabolic syndrome Hyperuricemia is also found to be associated with progressive renal dysfunction which is also proved in rat model experiments.GFR<60 ml and albuminuria are associated with metabolic syndrome for which the mechanism remains unclear. Obesity is mentioned as a causative factor. Obesity is a component of metabolic syndrome which is associated with causing renal dysfunction in various ways. Obesity causes glomerular sclerosis . It also causes fat deposition in renal medulla thereby tubular flow decreases resulting in volume overload and hypertension. Metabolic syndrome also causes release of cytokines causing endothelial dysfunction and oxidative stress. Dyslipidemia caused by raised uric acid levels also causes interstitial injury to the kidneys. Thus raised uric acid levels by causing metabolic syndrome contributes to development of Chronic Kidney Disease.

URIC ACID AND GOUT

Gout is a form of arthritis inflammatory in nature caused due to deposition of urate crystals within the joints and periarticular tissues. It occurs when high levels of uric acid remains in the serum without excretion which causes its crystallisation. Arthritis is an inflammatory response to the deposition of needle shaped crystals within the joint cavities and surrounding tissues. It is associated with tender, erythematous and swollen joints. Pain would be intense initially and later it persists for days to weeks. Person susceptible to the development of Gout are

- Obese
- Renal involvement
- Excess alcohol exposure
- Persons on diuretics
- Family h/o gout
- Exposure to lead

DIAGNOSIS can be confirmed by joint fluid aspirate and demonstration of urate crystals in the sample.Blood tests can be misleading since mostly during attacks of pain levels of uric acid remains within normal range and it is proposed that attack is precipitated due to sudden changes in levels causing crystallisation.Thus fluctuation in levels of uric acid is the key determinant in precipitating attacks.

TREATMENT is by drugs to lower the levels of uric acid. Gout cannot be cured but it can be better maintained with drugs. Patient has to be on drugs throughout his life which decreases their symptoms and risk of deformity or disability. Pain can be managed with NSAIDS which tend to reduce the inflammation and provide relief. Colchicine also reduces pain effectively if taken within 12 hours of initiation of attack and later to avoid future attacks it can be taken in lower doses. Life style modifications like Exercise and Diet adjustments also plays a role in prevention of future attacks. Patients are advised to have less purine containing foods and to avoid mushrooms, meat, asparagus and beans which has high purine content. Patients are also advised to stop alcohol as it raises the levels of uric acid and suggested to have plenty of water.If left untreated can lead to permanent damage of kidneys and joints.

There are atleast three inherited defects causing early development of Hyperuricemia and Gout:

- Glucose 6-phosphatase deficiency – VON GIERKE DISEASE
- Hypoxanthine –Guanine phosphoribosyl transferase deficiency(partial and severe) –LESCH NYHAN SYNDROME(severe)
- Raised 5’phosphoribosyl-1’-pyrophosphate synthetase activity

REDUCTION OF URIC ACID AND PROTECTION AGAINST STROKE

Drugs acting to reduce uric acid levels do so by inhibiting Xanthine oxidase or increasing excretion of uric acid like probenecid, sulfinpyrazone. Other drugs like losartan, fenofibrates, and statins also reduces uric acid levels.

The findings of LIFE study (Losartan Intervention for Endpoint Reduction in hypertension study) suggest that a decrease in Serum uric acid induced by losartan treatment attenuates CV risk, including stroke.[80]

Asterios Karagiannis et al[81] studied consecutive patients (n=435) presenting with ischemic stroke and intracerebral hemorrhage and concluded that Elevated levels of Serum uric acid are independently associated with an increased risk of early death in acute stroke.

Newman EJ, et al [82] studied a cohort of type 2 diabetes patients presenting to their unit with computed tomography-confirmed acute stroke. Concluded that elevated urate concentration is significantly and independently associated with increased risk of future vascular events in diabetic stroke patients. Further studies to elucidate the mechanism of this observation are required.

Christopher J. Weir, et al[52] studied 3731 patients with ischemic stroke or primary intracranial hemorrhage, and determined the association of urate level with 90-day placement (alive at home, good outcome; dead or living in care,

poor outcome) and concluded that independent of other prognostic factors, higher serum urate levels predicted poor outcome (dead or in care) and higher vascular events.

Antonio Cherubini et al[55] studied antioxidant profile in patients with acute ischemic stroke of recent onset (<24 hours) and concluded that patients with the worst early outcome (death or functional decline) had higher vitamin A and uric acid plasma levels and lower vitamin C levels and erythrocyte super oxide dismutase activity than those who remained functionally stable.

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA

Patients admitted as in patients at Department of Medicine, MADRAS MEDICAL COLLEGE & RAJIVGANDHI GOVT GENERAL HOSPITAL, CHENNAI with acute ischemic stroke during the3 period of APRIL 2016 to SEPTEMBER 2016 were taken for study considering the inclusion and exclusion criteria.

METHODS OF COLLECTION OF DATA

Information was collected from each patient through a printed proforma meeting the objectives of the study. Purpose of the study was carefully explained to patients and consent was taken.

Qualifying patients underwent detailed history, clinical examination, investigations and followed up for a period of 7 days. Serum uric acid was measured when the patient was admitted. Clinical outcome in terms of mortality and physical disability was assessed as per GLASGOW OUTCOME SCALE{GOS} which is:

1. Indicates death
2. Vegetative state(unable to interact with environment)
3. Severe disability(unable to live independently but can follow commands)
4. Moderate disability(capable of living independently but unable to return to work)

5. Mild or no disability(patient can return to work)

TYPE OF STUDY

Single centre observational prospective hospital based time bound study.

SAMPLING

Hospital statistics show that about 400 cases (average) of acute stroke were

Admitted to MMC&RGGGH in the year2016.the number of case after

INCLUSION CRITERIA

1. Patients who were admitted with first episode of acute ischemic stroke with CT evidence of infarction

EXCLUSION CRITERIA

1. Patients with subarachnoid hemorrhage, extra dural hemorrhage, sub dural hemorrhage and intra cerebral hemorrhage on CT.
2. Patients with previous history of TIA/RIND.
3. Patients with gout
4. Patients taking drugs causing hyperuricemia.
5. Patients who were alcoholics.

Such cases of acute ischemic stroke satisfying inclusion and exclusion criteria admitted in medical wards of MMC&RGGGH from March 2016 to August 2016 were taken for study as time bound study.

STATISTICAL ANALYSIS

SPSS statistical software was used to analyse data.MS office word and MS Excel programmes were used to make charts ,ratios

RESULTS

RESULTS

AGE DISTRIBUTION

Table 1: Age Distribution of Subjects

AGE_GROUP	FREQUENCY	PERCENT
40-50 years	18	30.0
3451-60 years	15	25.0
61-70 years	20	33.3
71-80 years	5	8.3
>80 years	2	3.3
Total	60	100.0

Descriptive Statistics	N	Minimum	Maximum	Mean	Std. Deviation
AGE_	60	40.00	84.00	59.2167	11.17365

In this prospective study, majority of stroke patients were between 61 to 70 years old(33%) whereas 40 to 50 years age group constituted 30% of study population.

Fig:1

Age Distribution

Thus in our study patients of 41 to 90 years were included and the observations made were analysed and depicted.

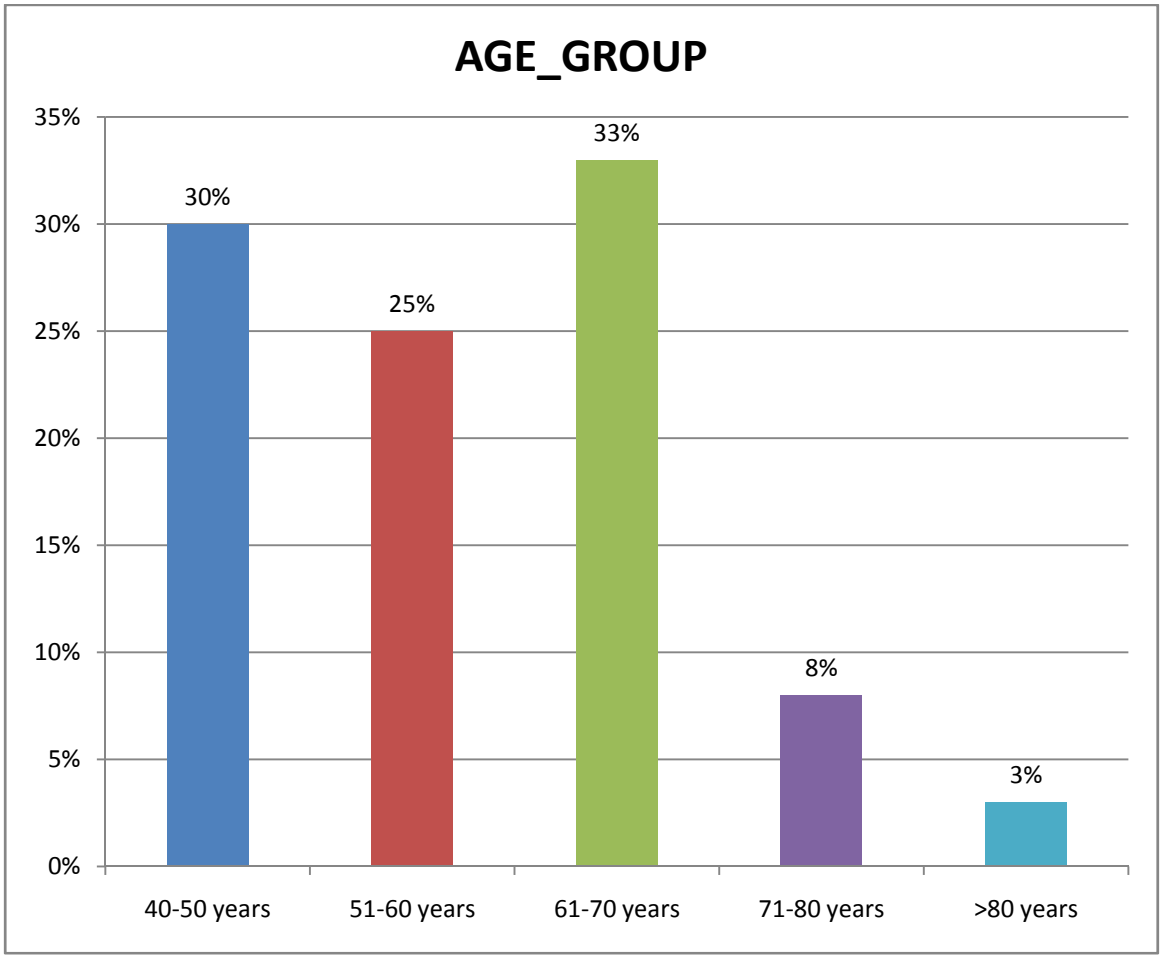


Table: 2

AGE DISTRIBUTION ACCORDING TO SEX

AGE_GROUP * SEX CROSSTABULATION			SEX		Total
			FEMALE	MALE	
Age_Group	40-50 years	Count	2	16	18
		% within SEX	33.3%	29.6%	30.0%
	51-60 years	Count	2	13	15
		% within SEX	33.3%	24.1%	25.0%
	61-70 years	Count	1	19	20
		% within SEX	16.7%	35.2%	33.3%
	71-80 years	Count	1	4	5

		% within SEX	16.7%	7.4%	8.3%
	>80 years	Count	0	2	2
		% within SEX	0.0%	3.7%	3.3%
Total		Count	6	54	60
		% within SEX	100.0%	100.0%	100.0%

Pearson Chi-Square =1.543p=.819

Majority of males are in 61 to 70 years age group.

Majority of females are in 41 to 60 years age group.

Fig:2

AGE DISTRIBUTION ACCORDING TO SEX

Hence there is no association found between sex of the patient and stroke incidence.

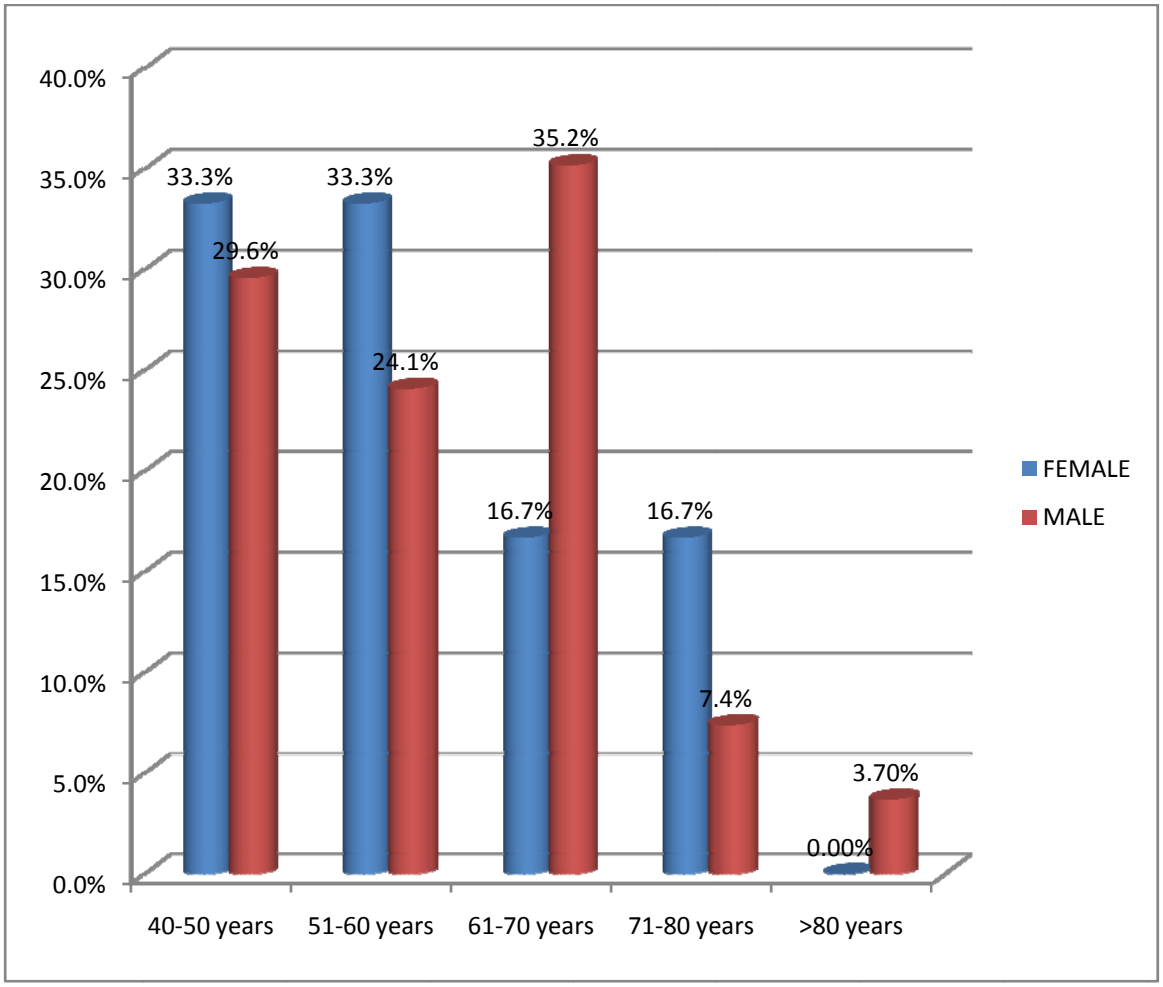


TABLE:3

RISK FACTORS

		N	Y
SHT	Count	18	42
	Table N %	30.0%	70.0%
D_M	Count	46	14
	Table N %	76.7%	23.3%
SMOKING	Count	53	7
	Table N %	88.3%	11.7%
DYSLIPIDEMIA	Count	53	7
	Table N %	88.3%	11.7%
CAD	Count	53	7
	Table N %	88.3%	11.7%

Hypertension constituted the major risk factor in this stroke population as 70% were hypertensive.

Diabetes Mellitus ranked second as a risk factor constituting 14% of the population.

Smoking ; Dyslipidemia ;Coronary Artery Disease constituted to 11.7% of the population each.

Fig:3

RISK FACTORS IN THE POPULATION

Thus Hypertension constituted the major risk factor in the population.

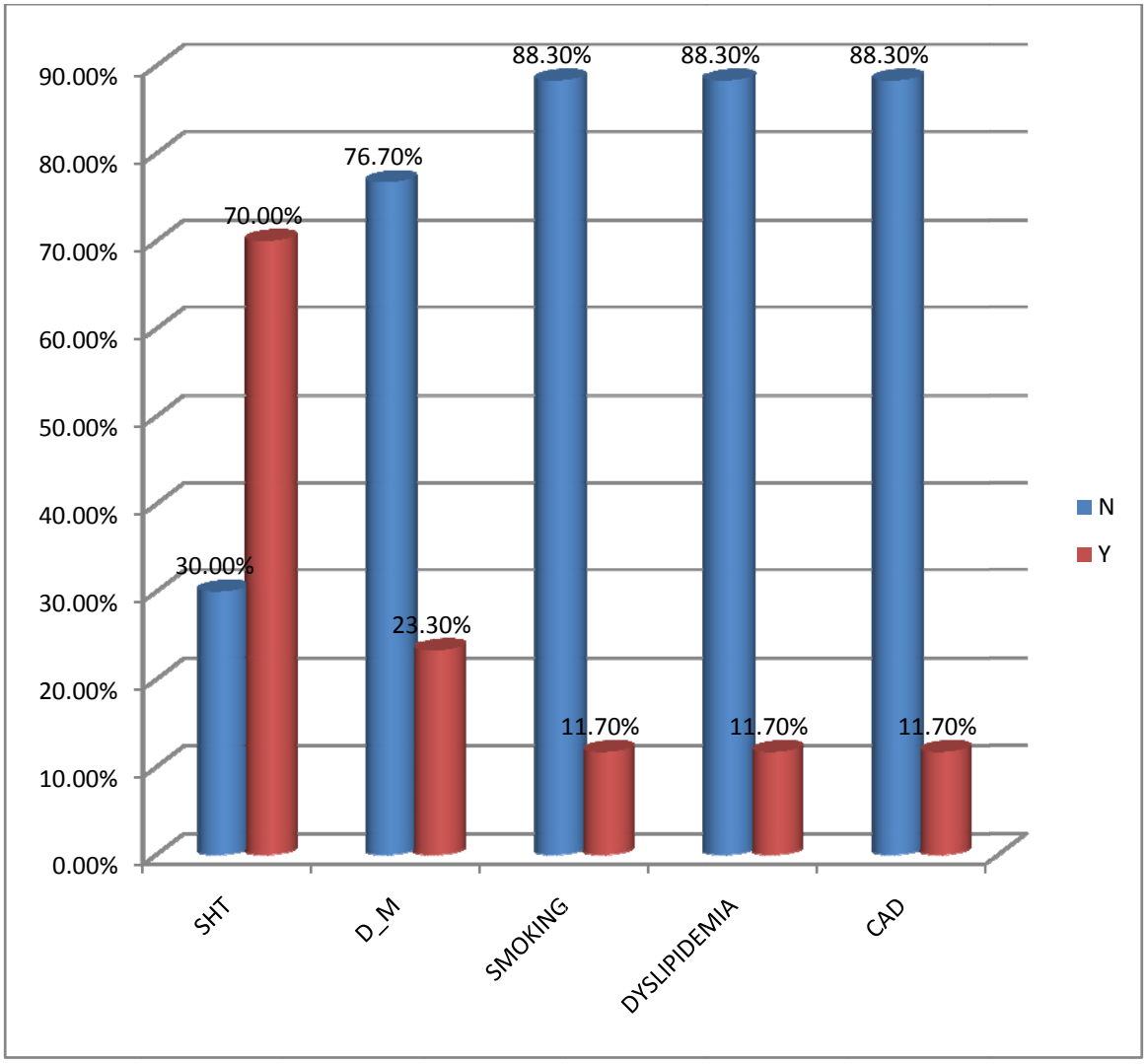


Table:4

RISK FACTORS ACCORDING TO GENDER OF THE PATIENT

		FEMALE	**MALE**
SHT	Count	4	38
	Table N %	6.7%	63.3%
D_M	Count	2	12
	Table N %	3.3%	20.0%
SMOKING	Count	0	7
	Table N %	0.0%	11.7%
DYSLIPIDEMIA	Count	2	5
	Table N %	3.3%	8.3%
CAD	Count	1	6
	Table N %	1.7%	10.0%

Hereby 63.3% of males are Hypertensives.

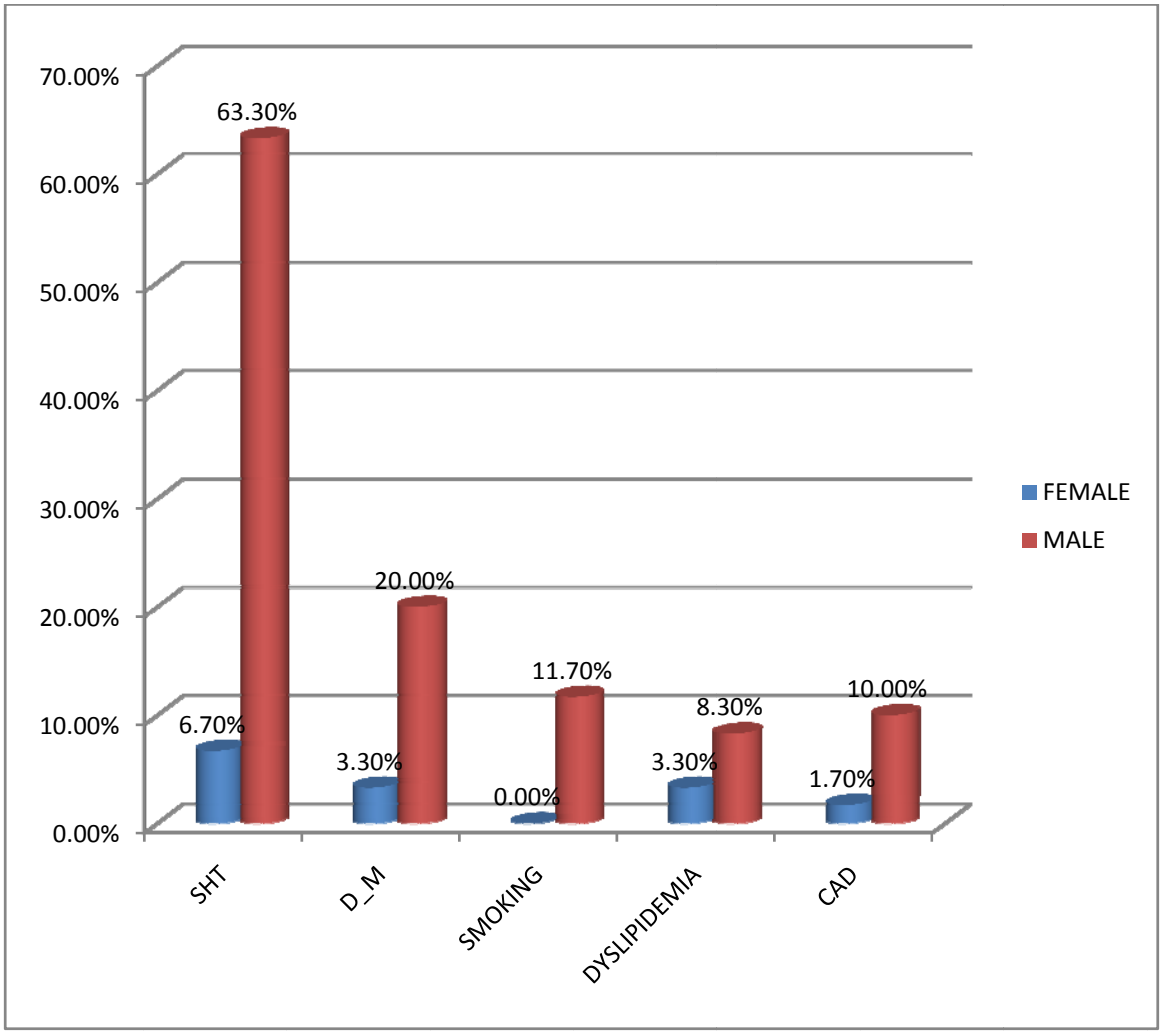
20% of males are Diabetics.

6.7% of females are Hypertensives.

Thus majority of males in this stroke population are Hypertensives.

Fig:4

RISK FACTORS ACCORDING TO GENDER OF THE PATIENT



Hypertension is the leading risk factor in both sexes of the study population.

TABLE:5

DISTRIBUTION OF URIC ACID LEVELS

URIC_ACID_CLASS	FREQUENCY	PERCENT
<5	7	11.7
5-7	12	20.0
>7	41	68.3
Total	60	100.0

Descriptive Statistics	N	Minimum	Maximum	Mean	Std. Deviation
URIC_ACID	60	3.50	14.00	8.4550	2.50054
Valid N (listwise)	60				

Mean serum uric acid value of study population is 8.45 mg/dl. Majority of subjects had high serum uric acid levels of >7 mg/dl (68.3%) whereas 20% had

Fig: 5

uric acid levels within the normal levels.

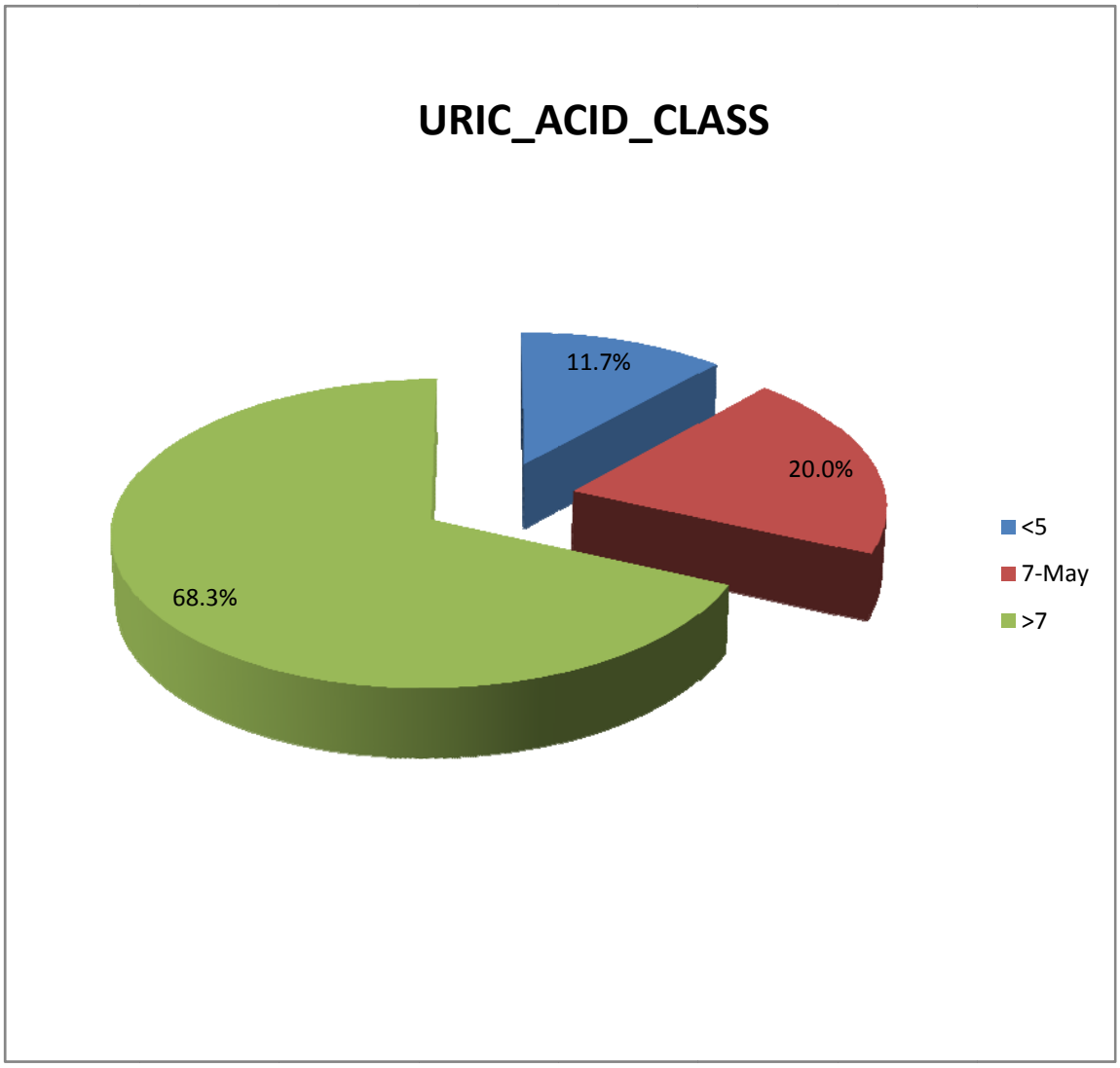


TABLE :6

URIC ACID AND GENDER

URIC_ACID_CLASS * SEX Crosstabulation			SEX		Total
			FEMALE	MALE	
URIC_ACID_CLASS	<5	Count	1	6	7
		% within SEX	16.7%	11.1%	11.7%
	5-7	Count	1	11	12
		% within SEX	16.7%	20.4%	20.0%
	>7	Count	4	37	41
		% within SEX	66.7%	68.5%	68.3%
Total		Count	6	54	60
		% within SEX	100.0%	100.0%	100.0%

Majority of males in the study population have high serum uric acid levels of >7mg/dl (68.5%).Majority of females also have high uric acid levels of>7mg/dl(66.7%)

Hence majority of subjects in both sexes havew high serum uric acid levels

Fig:6

URIC ACID AND GENDER

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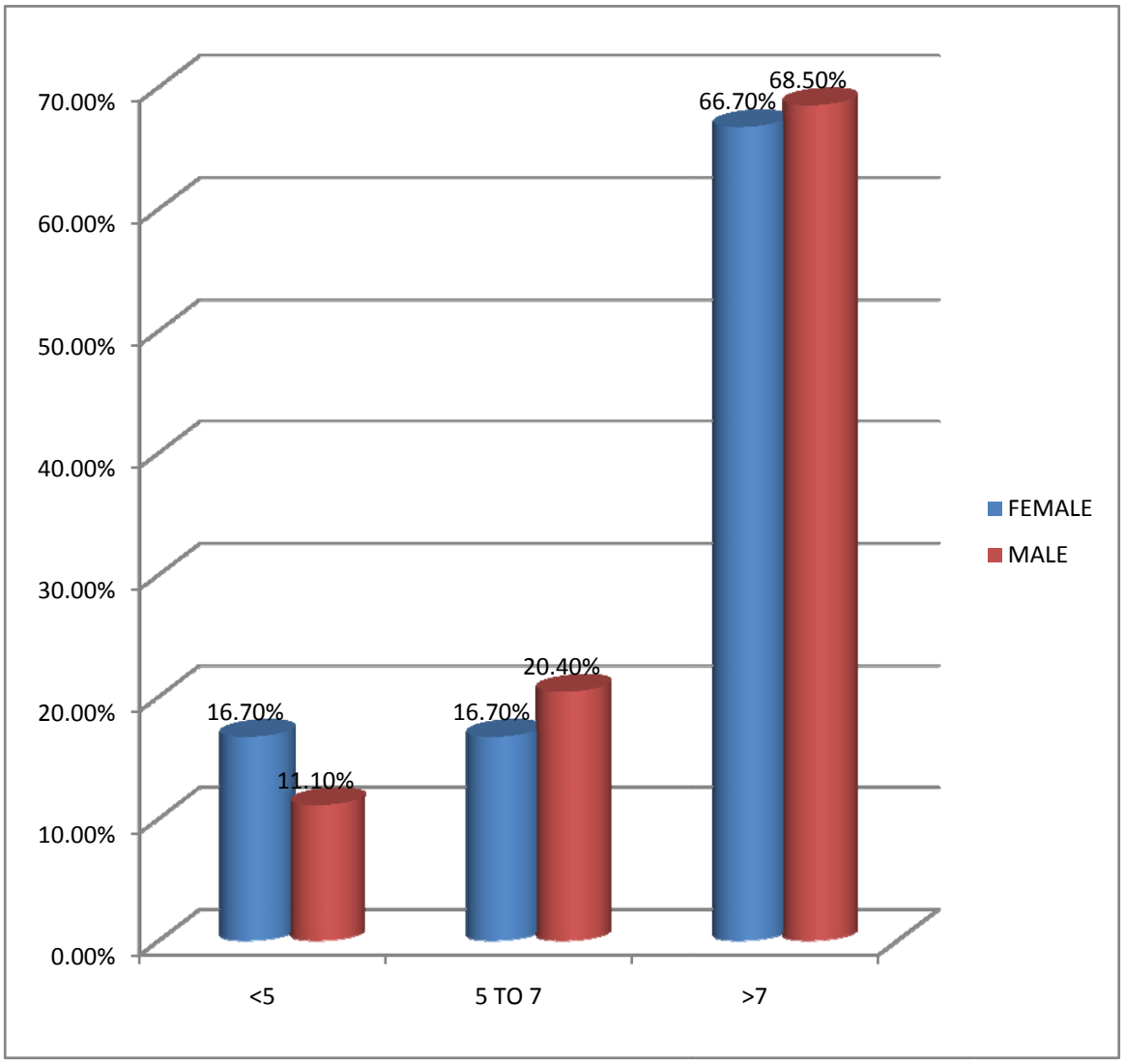

TABLE 7: DISTRIBUTION OF AGE GROUPS AND URIC ACID LEVELS

URIC_ACID_CLASS * age_group Crosstabulation			AGE_GROUP					Total
			40-50 years	51-60 years	61-70 years	71-80 years	>80 years	
URIC_ACID_CLASS	<5	Count	1	2	4	0	0	7
		% within age_group	5.6%	13.3%	20.0%	0.0%	0.0%	11.7%
	5-7	Count	3	3	4	2	0	12
		% within age_group	16.7%	20.0%	20.0%	40.0%	0.0%	20.0%
	>7	Count	14	10	12	3	2	41
		% within age_group	77.8%	66.7%	60.0%	60.0%	100.0%	68.3%
Total		Count	18	15	20	5	2	60
		% within age_group	100.0 %	100.0 %	100.0 %	100.0%	100.0%	100.0 %

Most of the persons with high uric acid levels are in the age group 40 to 50 .

Next is 61 to 70 years group and 51 to 60 years group.

Fig:7

DISTRIBUTION OF AGE GROUP AND URIC ACID LEVELS

Hence there is no linear increase found in levels of uric acid as age advances

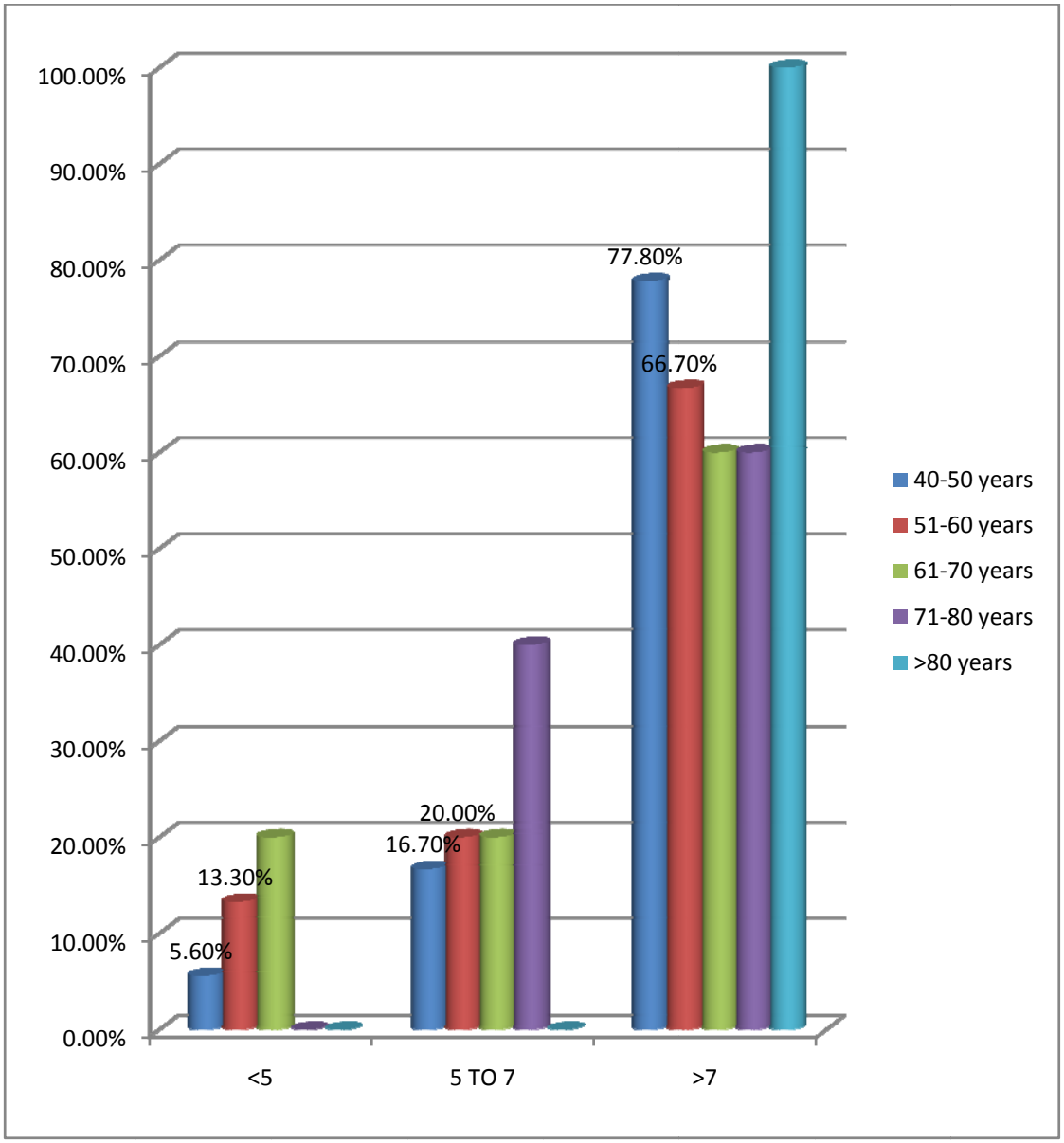

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TABLE:8

AGE GROUPS WITH MEAN URIC ACID LEVELS

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean	
					Lower Bound	Upper Bound
40-50 years	18	8.8333	2.16251	.50971	7.7579	9.9087
51-60 years	15	8.6733	3.02170	.78020	7.0000	10.3467
61-70 years	20	7.6450	2.23477	.49971	6.5991	8.6909
71-80 years	5	8.7000	2.16795	.96954	6.0081	11.3919
>80 years	2	10.9000	4.38406	3.10000	-28.4892	50.2892
Total	60	8.4550	2.50054	.32282	7.8090	9.1010

F=1.159 P=0.339 NON SIGNIFICANT

There is no significant difference between age and serum uric acid levels.

TABLE :9

GENDER WITH MEAN URIC ACID

	SEX	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
URIC_ACID	MALE	54	8.5333	2.50742	.34122
	FEMALE	6	7.7500	2.54460	1.03883

t=0.725 P=0.471 NON SIGNIFICANT

There is no significant difference in males and females in uric acid levels.

TABLE :10

HYPERTENSION WITH MEAN URIC ACID

Group Statistics							
SHT		**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**t value**	**p value**
URIC_ACID	PRESENT	42	8.5762	2.50734	.38689	.570	.571
	ABSENT	18	8.1722	2.53326	.59710		

There is no significant difference between serum uric acid levels and hypertension.

TABLE:11

DIABETES WITH MEAN URIC ACID

D_M		N	Mean	Std. Deviation	Std. Error Mean	t value	p value
URIC_ACID	PRESENT	14	8.2571	2.87903	.76945	-.336	.738
	ABSENT	46	8.5152	2.40573	.35471		

There is no significant difference between diabetes and serum uric acid.

TABLE:12

SMOKING WITH MEAN SERUM URIC ACID

SMOKING		**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**T value**	**p value**
URIC_ACID	PRESENT	7	6.9000	2.06236	.77950	-1.783	.080
	ABSENT	53	8.6604	2.49702	.34299		

There is no significant difference between smoking and serum uric acid.

TABLE:13

DYSLIPIDEMIA WITH MEAN URIC ACID

DYSLIPIDEMIA		**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**	**t value**	**p value**
URIC_ACID	PRESENT	7	8.2143	1.91174	.72257	-.269	.789
	ABSENT	53	8.4868	2.58145	.35459		

There is no significant difference between dyslipidemia and serum uric acid.

TABLE:14

CORONARY ARTERY DISEASE WITH MEAN URIC ACID

CAD		N	Mean	Std. Deviation	Std. Error Mean	t value	p value
URIC_ACID	PRESENT	7	8.1143	3.12380	1.18068	-.381	.705
	ABSENT	53	8.5000	2.43942	.33508		

There is no significant difference between coronary artery disease and uric acid.

TABLE:15

ARTERIAL TERRITORY WITH MEAN URIC ACID

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean	
					Lower Bound	Upper Bound
MCA	37	8.8784	2.71336	.44607	7.9737	9.7831
PCA	5	9.0000	1.27475	.57009	7.4172	10.5828
ACA	8	7.2250	1.41093	.49884	6.0454	8.4046
PCA+MCA	7	7.1429	1.90863	.72139	5.3777	8.9080
ACA+MCA	3	8.6667	3.81881	2.20479	-.8198	18.1531
Total	60	8.4550	2.50054	.32282	7.8090	9.1010

F=1.324 P=0.272 NON SIGNIFICANT. There is no significant difference.

TABLE:16

COMPARISON OF VASCULAR TERRITORY WITH URIC ACID

CT_EVIDENCETERRITORY * URIC_ACID_CLASS Crosstabulation			**URIC_ACID_CLASS**			**Total**
			<5	**5-7**	**>7**	
CT_Evidenceterritory	MCA	Count	3	8	26	37
		% within Uric_Acid_Class	42.9%	66.7%	63.4%	61.7%
	PCA	Count	0	0	5	5
		% within Uric_Acid_Class	0.0%	0.0%	12.2%	8.3%
	ACA	Count	1	3	4	8
		% within Uric_Acid_Class	14.3%	25.0%	9.8%	13.3%
	PCA+M	Count	2	1	4	7

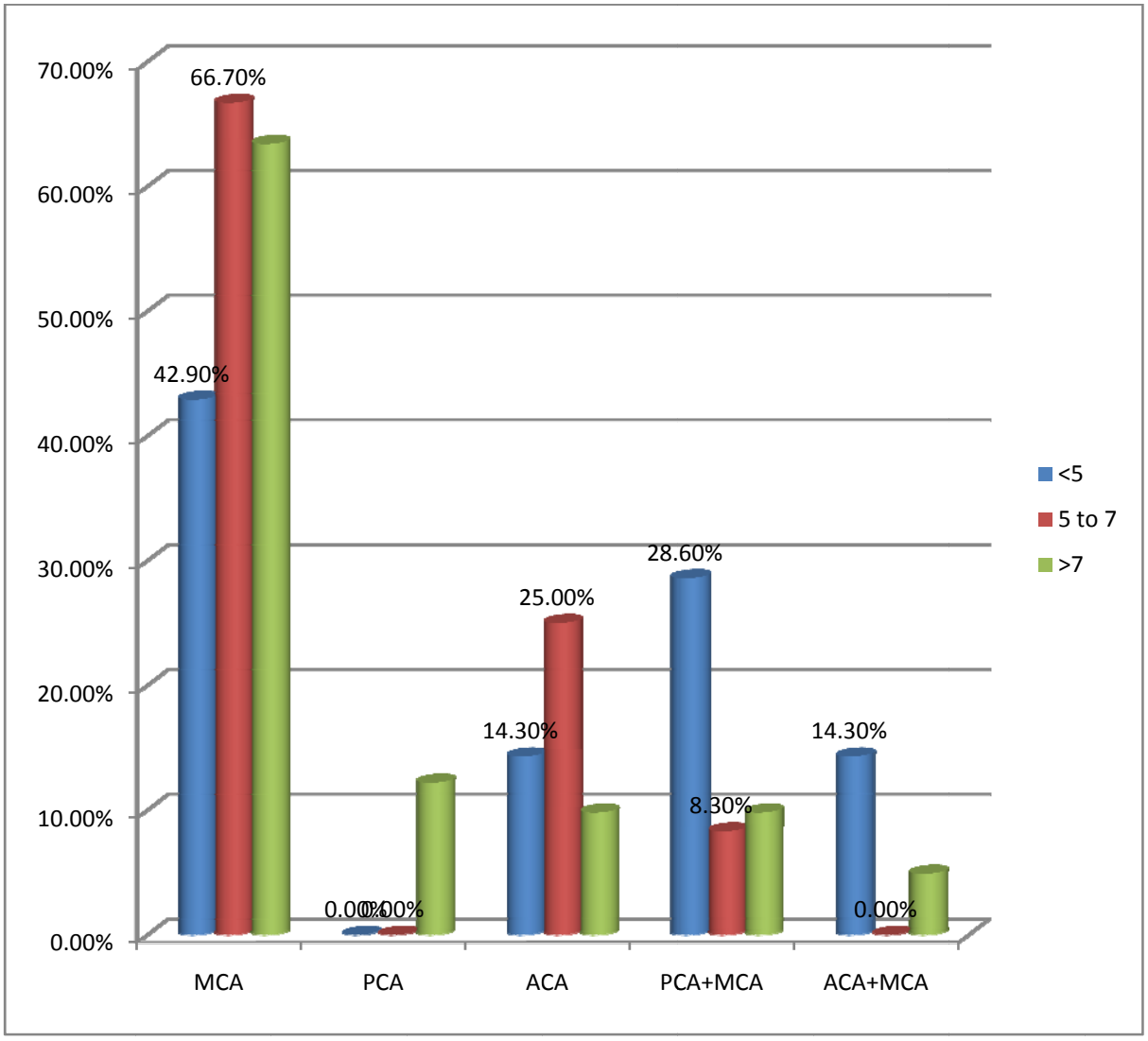
	CA	% within URIC_ACID_CLASS	28.6%	8.3%	9.8%	11.7%
	ACA+MCA	Count	1	0	2	3
		% within URIC_ACID_CLASS	14.3%	0.0%	4.9%	5.0%
Total		Count	7	12	41	60
		% within URIC_ACID_CLASS	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square =8.176 P =0.416 NON SIGNIFICANT

Hereby vascular territory involved was compared with mean serum uric acid and it was found that persons with MCA territory involvement had high uric acid levels.

Fig:8

VASCULAR TERRITORY WITH URIC ACID



Though MCA territory involved patients had high uric acid levels it was not statistically significant.

TABLE:17

HYPERTENSION AND GLASGOW OUTCOME SCALE

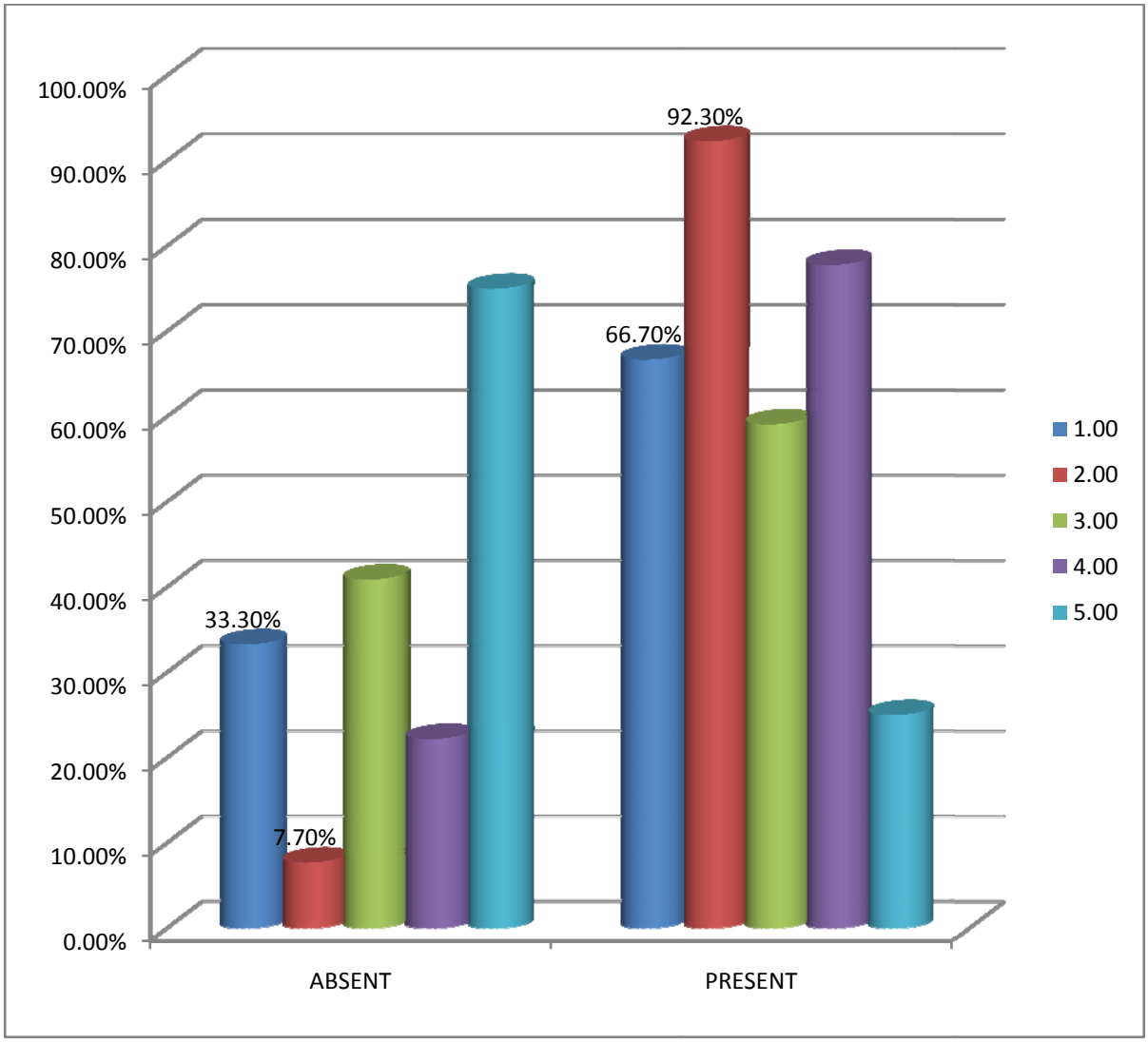
CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
			1.00	**2.00**	**3.00**	**4.00**	**5.00**	
SHT	ABSENT	Count	1	1	9	4	3	18
		% within GLASGOW_OUTCOME_SCALE	33.3%	7.7%	40.9 %	22.2 %	75.0 %	30.0 %
	PRESENT	Count	2	12	13	14	1	42
		% within GLASGOW_OUTCOME_SCALE	66.7%	92.3 %	59.1 %	77.8 %	25.0 %	70.0 %
Total		Count	3	13	22	18	4	60
		% within GLASGOW_OUTCOME_SCALE	100.0 %	100. 0%	100. 0%	100. 0%	100. 0%	100. 0%

Pearson Chi Square=8.719 P=0.069

Among 60 persons 42 had hypertension(30%).Among them 12 persons had poor GOS OF 2.0

FIG:9

HYPERTENSION AND GLASGOW OUTCOME SCALE



Hereby in GCS 2.0 category hypertension was found in 92.3% of subjects.

TABLE:18

DIABETES MELLITUS AND GLASGOW OUTCOME SCALE

CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
			1.00	2.00	3.00	4.00	5.00	
D_M	ABSENT	Count	2	10	17	14	3	46
		% within GLASGOW_OUTCOME_SCALE	66.7%	76.9%	77.3%	77.8%	75.0%	76.7%
	PRESENT	Count	1	3	5	4	1	14
		% within GLASGOW_OUTCOME_SCALE	33.3%	23.1%	22.7%	22.2%	25.0%	23.3%
Total		Count	3	13	22	18	4	60
		% within GLASGOW_OUTCOME_SCALE	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square=0.191 P =0.996

Among 60 persons 14 had Diabetes. But no significant association was found between Diabetes and Glasgow Outcome Scale.

TABLE:19

SMOKING AND GLASGOW OUTCOME SCALE

CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
			1.00	2.00	3.00	4.00	5.00	
SMOKING	ABSENT	Count	3	13	18	16	3	53
		% within GLASGOW_OUTCOME_SCALE	100.0%	100.0%	81.8%	88.9%	75.0%	88.3%
	PRESENT	Count	0	0	4	2	1	7
		% within GLASGOW_OUTCOME_SCALE	0.0%	0.0%	18.2%	11.1%	25.0%	11.7%
Total		Count	3	13	22	18	4	60
		% within GLASGOW_OUTCOME_SCALE	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square=3.715 P=0.446

Among 60 persons 7 were smokers.

But there is no significant association between Smoking and Glasgow Outcome Scale.

TABLE :20

DYSLIPIDEMIA AND GLASGOW OUTCOME SCALE

CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
				2.00	3.00	4.00	5.00	
Dyslipidemia	Absent	Count	3	11	20	16	3	53
		% within Glasgow_ Outcome_Scale	100.0 %	84.6%	90.9%	88.9%	75.0 %	88.3%
	Present	Count	0	2	2	2	1	7
		% within Glasgow_ Outcome_Scale	0%	15.4%	9.1%	11.1%	25.0 %	11.7%
Total		Count	3	13	22	18	4	60
		% within Glasgow_Outcome_Scale	100.0 %	100.0 %	100.0 %	100.0 %	100.0 %	100.0 %

Pearson Chi Square =1.408 P=O.843

Among 60 persons 7 had dyslipidemia.

But there is no significant association between Dyslipidemia and GOS.

TABLE:21

CORONARY ARTERY DISEASE AND GLASGOW OUTCOME SCALE

CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
			1.00	2.00	3.00	4.00	5.00	
CAD	ABSENT	Count	2	12	19	17	3	53
		% within GLASGOW_OUTCOME_SCALE	66.7%	92.3%	86.4%	94.4%	75.0%	88.3%
	PRESENT	Count	1	1	3	1	1	7
		% within GLASGOW_OUTCOME_SCALE	33.3%	7.7%	13.6%	5.6%	25.0%	11.7%
Total		Count	3	13	22	18	4	60
		% within GLASGOW_OUTCOME_SCALE	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square =2.991 P=0.559

Among 60 persons 7 were having Coronary Artery Disease.

But there is no significant association between Coronary Artery Disease and GOS.

TABLE:22

TERRITORY INVOLVEMENT AND GLASGOW OUTCOME SCALE

CROSSTAB			Glasgow_Outcome_Scale					Total
			1.00	2.00	3.00	4.00	5.00	
CT_EVIDENCETERRITORY	MCA	Count	3	11	11	9	3	37
		%	100.0%	84.6%	50.0%	50.0%	75.0%	61.7%
	PCA	Count	0	1	3	1	0	5
		%	0.0%	7.7%	13.6%	5.6%	0.0%	8.3%
	ACA	Count	0	0	3	4	1	8
		%	0.0%	0.0%	13.6%	22.2%	25.0%	13.3%
	PCA+MCA	Count	0	0	5	2	0	7
		%	0.0%	0.0%	22.7%	11.1%	0.0%	11.7%
	ACA+MCA	Count	0	1	0	2	0	3
		%	0.0%	7.7%	0.0%	11.1%	0.0%	5.0%
Total		Count	3	13	22	18	4	60
		%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square=15.560 P=0.484

Among 60 persons 37 were having MCA infarct and persons with MCA infarct were prone for poor GOS score.

But it was not statistically significant.

TABLE:23

AGE GROUP AND GLASGOW OUTCOME SCALE

CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
			1.00	2.00	3.00	4.00	5.00	
SEX	MALE	Count	3	12	19	17	3	54
		%	100.0%	92.3%	86.4%	94.4%	75.0%	90.0%
	FEMALE	Count	0	1	3	1	1	6
		%	0.0%	7.7%	13.6%	5.6%	25.0%	10.0%
Total		Count	Count	13	22	18	4	60
		% within GLASGOW_OUTCOME_SCALE	%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square=22.522 P=0.127

Among 60 persons 20 were in age group of 61 to 70 years, next is 40 to 50 years and 50 to 60 years.

There is no statistical relationship between Age group and GOS.

TABLE:24

SEX DIFFERENCE AND GLASGOW OUTCOME SCALE

CROSSTAB			GLASGOW_OUTCOME_SCALE					Total
			1.00	2.00	3.00	4.00	5.00	
Age_group	40-50 years	Count	2	2	9	4	1	18
		%	66.7%	15.4%	40.9%	22.2%	25.0%	30.0%
	51-60 years	Count	0	6	3	4	2	15
		%	0.0%	46.2%	13.6%	22.2%	50.0%	25.0%
	61-70 years	Count	0	4	6	9	1	20
		%	0.0%	30.8%	27.3%	50.0%	25.0%	33.3%
	71-80 years	Count	0	1	3	1	0	5
		%	0.0%	7.7%	13.6%	5.6%	0.0%	8.3%
	>80 years	Count	1	0	1	0	0	2
		%	33.3%	0.0%	4.5%	0.0%	0.0%	3.3%
Total		Count	Count	13	22	18	4	60
		%	%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi Square=2.129 P =0.712

Among 60 persons 54 were males .

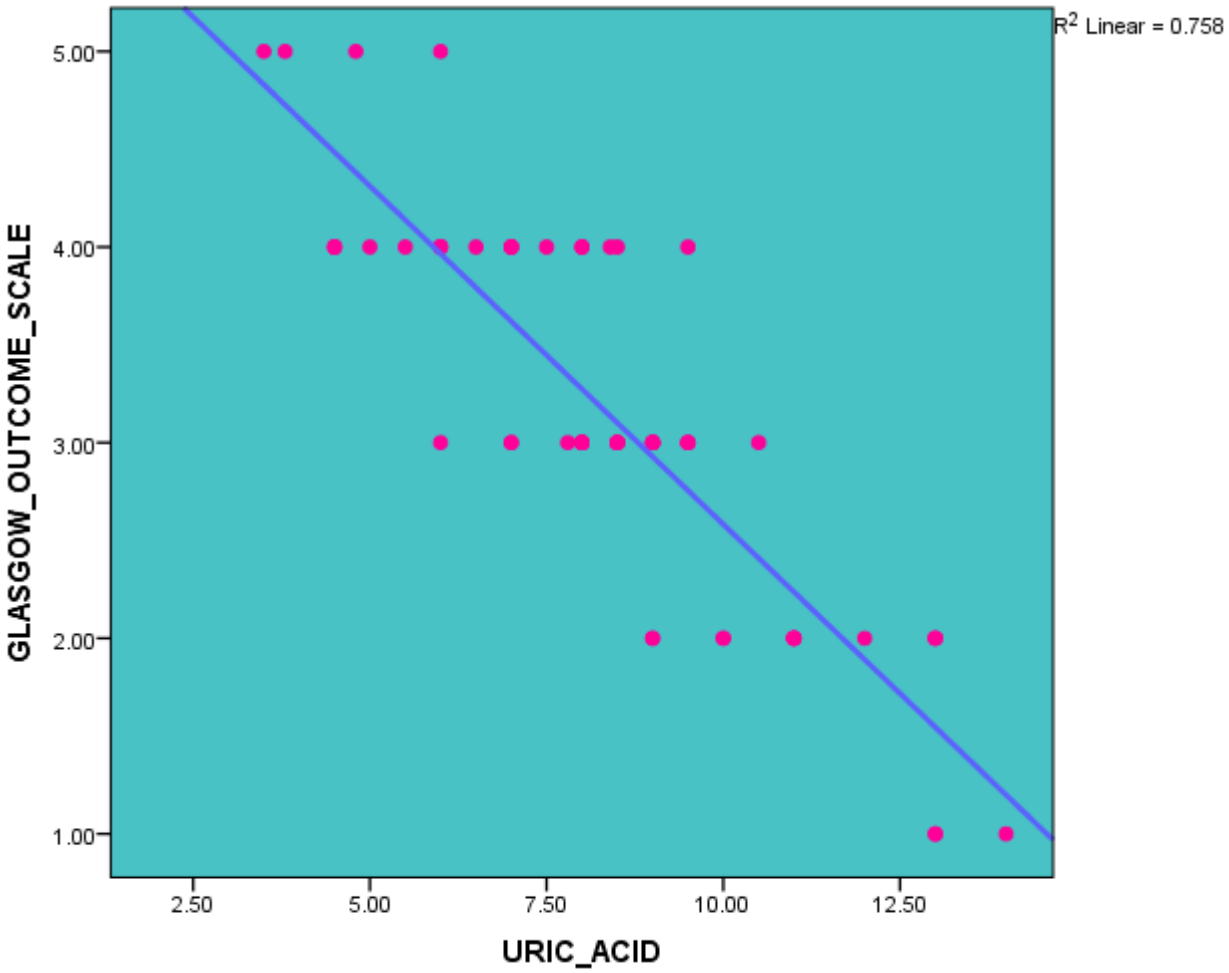
No significant relationship exists between sex and GOS.

TABLE :25

CORRELATION BETWEEN SERUM URIC ACID LEVEL AND GOS

BY SPEARMAN'S METHOD

			URIC_ACID
Spearman's rho	GLASGOW_OUTCOME_SCALE	Correlation Coefficient	-.856**
		Sig. (2-tailed)	.000
		N	60



From the above graph it shows that as Serum uric acid level raises GOS score decreases . Thus the outcome of patients with high serum uric acid levels is poor compared to normal or low uric acid levels.

Hence there is a Negative Correlation between Serum uric acid levels and GOS.

Model Summary[b]									
Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	.871[a]	.758	.754	.49233	.758	182.038	1	58	.000
a. Predictors: (Constant), URIC_ACID									
b. Dependent Variable: GLASGOW_OUTCOME_SCALE									

Coefficients[a]								
Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.	95.0% Confidence Interval for B	
		B	Std. Error	Beta			Lower Bound	Upper Bound
1	(Constant)	6.041	.226		26.746	.000	5.589	6.493
	URIC_ACID	-.346	.026	-.871	-13.492	.000	-.397	-.295
a. Dependent Variable: GLASGOW_OUTCOME_SCALE								

With above inferences GOS can be estimated from Serum uric acid levels by the Equation:

GOS =-0.346(URIC ACID) + 6.041

Eg:

If uric acid level is 12 ,then as per equation -4.152+6.041, GOS is 2.

Hereby we can predict the GOS by using Serum uric acid levels.

TABLE:26

URIC ACID AND GLASGOW OUTCOME SCALE

gos * URIC_ACID_CLASS Crosstabulation						
			URIC_ACID_CLASS			Total
			<5	5-7	>7	
Gos	bad (1-3)	Count	0	3	35	38
		%	0.0%	7.89%	92.11%	63.3%
	good (4-5)	Count	7	9	6	22
		%	32%	41%	27%	36.7%
Total		Count	7	12	41	60
		% within URIC_ACID_CLASS	100.0%	100.0%	100.0%	100.0%

overall 38 (63%) were having bad outcome .among 38 patients 35(92%) were having high SUA

Thus by our study most of the population have poor GOS and among them 92% have high serum uric acid levels.

It is also proved statistically significant and there exists a negative correlation between serum uric acid levels and GOS Score.

DISCUSSION

DISCUSSION

RISK FACTORS

The risk factors considered in this study are HYPERTENSION, DIABETES MELLITUS, CORONARY ARTERY DISEASE, SMOKING and DYSLIPIDEMIA.

Among the risk factors ranking first is Hyperetension contributing about 70% of the study population which is followed by Diabetes constituting around 14% of the population. Next ranking risk factors are Smoking, Coronary artery disease and Dyslipidemia constituting 11.7% each.

63% of total male population are Hypertensives.4% of females are Hypertensives. 20% of males are Diabetics.

STUDIES ON INDIVIDUAL RISK FACTORS

Studies which give the prevalence of various risk factors are mentioned below: SERUM URIC ACID AND HYPERTENSION

Study by Jae Joong Lee(Lee et al.2015), mentioned that in males of less than 40 years uric acid association was strong with respect to both Systolic and Diastolic pressures. This association was found to be strong in females than males. But it was not significant in other age groups .

Study by R.J.Johnson 2003 dealt with in detail the effects of Hyperuricemia on increased cardiovascular morbidity and pathogenesis of

Hypertension by renal effects. Serum uric acid was high in males and post menopausal females with increased cardio vascular risk.(estrogen is uricosuric).Serum uric acid is proved to be associated with endothelial dysfunction.

Wang et al proved the association between high uric acid levels and systolic BP.

Also Chammaro et al proved significant association between BP and Raised uric acid levels.

In our study totally 67% are hypertensives. In both males and females Hypertension constitutes the major existing risk factor. But it was not statistically significant. Though in all other studies Hypertension is associated with increased levels of uric acid ,in our study Hypertension and Raised uric acid levels are not statistically significant.

DIABETES MELLITUS

Study by Lehto 1998, in Diabetic patients high uric acid levels are significantly associated with stroke incidence and also fatality was proved to be on the higher side.

In NHANES I epidemiologic study,1971-1992 it suggests an independent association between raised uric acid levels and increased risk of obesity, dyslipidemia and hypertension. Hyperuricemia is also found to be associated with Insulin Resistance.

Study by Dehghan 2008, suggests that serum uric acid is an independent risk factor associated with Diabetes.

Study by Satoru Kodama 2009,by Meta Analysis proved that Serum uric acid level is positively associated with Diabetes excluding other variables.

In our study , 23% were Diabetics thus constituting second most associated risk factor. Though above said studies proved an association between Hyperuricemia and Diabetes there was no statistically significant association between them in our study.

SMOKING

Study by Chammaro et al found no significant association between smoking and high uric acid levels.

In our study,11% were smokers and there was no statistically significant association between smokers and high serum uric acid levels.

DYSLIPIDEMIA

GREACE study 2004, suggests that serum uric acid levels are an independent risk factor for CAD. Hence when started on statin therapy it reduces uric acid levels thereby reducing overall future risk.

Study by Tsan Yang 2012, suggests an association between Hyperuricemia and Metabolic syndrome which is more common in females. Sex related association is yet to be studied.

Study by Ishizaka 2005, suggests that the prevalence of Metabolic syndrome showed gradual increase in incidence as uric acid levels rise.

In our study 11% had Dyslipidemia and there was no statistical significant association between them.

CORONARY ARTERY DISEASE (CAD)

Study by Christopher Bickel 2002,suggests that serum uric acid raised levels are associated with increased mortality in angiographically proved CAD patients.

The Rotterdam study by J Bos,in his follow up study for 8 years suggests that Hyperuricemia is associated with increased risk of Myocardial Infarction and Stroke.

Framingham study by F.N..BRAND, suggests that Hyperuricemia is significantly associated with CAD.

In our study,11% had CAD and it was not in significant association with Hyperuricemia.

PROGNOSIS OF STROKE PATIENTS AND URIC ACID LEVELS

In various studies mentioned above there exists independent association between Hyperuricemia and CAD, DYSLIPIDEMIA, HYPERTENSION, DIABETES MELLITUS and METABOLIC SYNDROME.

URIC ACID is considered to be also an Anti oxidant but which is proved to have pro oxidant effect in absence of other anti oxidants like Ascorbate.

Uric acid causes Endothelial Dysfunction and also decreases the levels of Nitric oxide which contributes the major pathophysiology for vascular events.

Hyperuricemia is found to increase the mortality in Diabetic Stroke patients and in aged population incidence of fatal stroke is increased.

Uric acid elevated levels are significantly associated with atherosclerosis and Dyslipidemia. Uric acid causes peroxidation of LDL and increases the production of oxygen free radicals. It also increases thrombus formation by increasing platelet aggregation. It is also proved that increased levels of uric acid crystals are found in atheromatous plaques of patients with high uric acid levels. According to Weir et al uric acid was proved to be a predictor of poor outcome in stroke patients. Three times risk is found to be associated with high uric acid levels.

Muir study suggests that Allopurinol which is used to treat Hyperuricemia decreases the levels of both uric acid and pro inflammatory markers. Hence it can be given to reduce future incidence of vascular events.

Karagiannis study mentioned an independent association between raised uric acid levels and stroke based earlier death.

European Heart Journal mentions that after an episode of stroke, uric acid levels can be used to assess the risk of cardiac deaths in those patients. It suggests that high Urate levels were associated with increased risk of cardiac death in future.

Khan et al proved the association between high uric acid levels and arterial stiffness. Hereby Hyperuricemics who are stroke survivors are prone for developing stiff vessels in near future.

In our study among 60 persons with stroke, 38 were having bad outcome (63%).Among them 35 were having high uric acid levels(92%).Hence our study suggests a significant association between high uric acid levels and poor GOS. There is no significant association between GOS in stroke patients and the associated risk factors like Smoking, Dyslipidemia and CAD in our study.

Also by Spearman”s correlation method ,it was found that as uric acid levels rise GOS score decreases. It was proved in the graph above. There exists a negative correlation between uric acid levels and GOS.

Thus studies around the world suggests association of raised uric acid levels with increased incidence of Diabetes, Dyslipidemia and Metabolic syndrome.

SUMMARY

SUMMARY

Uric acid is well known for its anti oxidant properties and free radical scavenging properties. It is also proved that in the absence of other anti oxidants like ascorbate it behaves in an opposite manner contributing to pro oxidant properties. Hence in our study we were looking at incidence of stroke patients with high uric acid levels and prognosis of those with higher values by assessing the GOS.

In our study of 60 patients, males were predominant in number. Age difference was not there between both sexes. Other risk factors like Dyslipidemia, Smoking, CAD and Hypertension were also taken into account. In our study population Mean Uric Acid level was found to be 8.45mg/dl. Majority of subjects had higher levels of >7mg/dl whereas 20% had normal uric acid levels.

In our study 38 persons were having poor outcome(63%).Among the 38 persons 35 were having high uric acid levels(92%).Hence by our study there exists a significant statistical association between serum uric acid level and GOS at the end of seven days of observation. Both were in negative correlation proved by Spearman’s correlation method.

CONCLUSION

CONCLUSION

Our study of 60 persons included persons suffering from ischemic stroke at presentation after fulfilling the exclusion and inclusion criteria mentioned above. Other risk factors like Age, Dyslipidemia, Smoking, Diabetes, CAD and Sex were taken into consideration. Among these most of the persons were of male sex and most of them were Hypertensives. But there was no statistical significant association between Male sex, Hypertension and Raised uric acid levels.

In our study 63% had poor outcome and among them 92% were having high uric acid levels. Based on our study it can be said that uric acid levels are used to prognosticate the stroke patients on their outcome basis at the end of seven days. Uric acid lowering drugs as a therapeutic option in stroke patients is in need of future research.

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PROFORMA

PROFORMA

"A STUDY OF SERUM URIC ACID AT PRESENTATION AS AN INDICATOR OF OUTCOME AMONG ACUTE ISCHEMIC STROKE PATIENTS"

NAME **I.P NO.**

AGE **OCCUPATION**

SEX **ADDRESS**

DATE OF ADMISSION

SOCIOECONOMIC STATUS

CHIEF COMPLAINTS ;

1.

2.

3.

4.

Seizures: Y / N

ICT Features; Y / N

H / O LOC : Y / N

h/s/o higher functions abnormality :

h/s/o cranial nerve lesions:

h/s/o motor system abnormalities:

h/s/o sensory involvement:

PAST HISTORY;

HTN- y/n DM – y/n

CAD- y/n CVA- y/n

TIA- y/n Hyperlipidemia – y/n

GOUT- y/n

PERSONAL HISTORY

SMOKING- Y/N ALCOHOLISM;- Y/N

DIET- Veg/ Non veg/ mixed

FAMILY HISTORY;

DM- ; HTN ;

CAD ; TIA ;

CVA ; GOUT

GENERAL PHYSICAL EXAMINATION;

Sensorium	posture
Built	Nourishment
Spine	skull

VITALS;

PULSE	TEMP
B.P	JVP
R.R	

SYSTEMIC EXAMINATION;

CENTRAL NERVOUS SYSTEM-

HIGHER MENTAL FUNCTIONS;

Consciousness;

Memory;

Speech

Behaivour

CRANIAL NERVES EXAMINATION

	RT	LT
OLFACTORY N		
OPTIC N		
Visual acuity		
Visual field		
Colour vision		
Fundoscopy		
III,IV,VI nerves		
EOM		
Conjugate movements		
Saccades		
Ptosis- Y/N		
Diplopia- Y/N		
PUPILS		
Size		
Shape		
Light reflex		
Accommodation reflex		

TRIGEMINAL N

Motor

Sensory

Jaw jerk

Corneal reflex

FACIAL N

Face appearance

Eye closure

Forehead wrinkling

Taste sensation

VESTIBULOCOCHLEAR N

Rinne’s

Weber’s

Auditory inattention

IX, X Nerves

Palatal movements

Gag reflex

SPINAL ACCESSORY N

Sternomastoid

Trapezius

HYPOGLOSSAL

Tongue movements

Wasting

Fasciculations

Involuntary movements

SIGNS OF MENINGEAL IRRITATION;

Neck stiffness

Kernig’s sign

MOTOR SYSTEM;

UPPER LIMB

Nutrition

Tone

Power

Co-ordination

LOWER LIMB

Nutrition

Tone

Power

Co-ordination

SENSORY SYSTEM

Superficial

Touch

Pain

Temp

Deep

Pain

Vibration

Position

Cortical sensations

REFLEXES

Superficial

Abdominal

Plantars

Deep

Biceps

Triceps

Supinator

Knee

Ankle

RHOMBERG'S TEST

GAIT

CEREBELLAR SYSTEM;

AUTONOMIC FUNCTION

OTHER SYSTEMS ;

(a CVS :

(b) RS :

(c) P / A:

INVESTIGATIONS;

1. COMPLETE HEMOGRAM

2. B .urea

S.creatinine

3. Blood : Sugar- mgs/dl

4. Lipid profile: TCL

LDL

HDL

TG

5. Serum uric acid-

6. ECG in all leads –

7. CT Brain -

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Sankara Avudayappan.A.P.
Post Graduate in M.D. (General Medicine)
Institute of Internal Medicine
Madras Medical College
Chennai 600 003

Dear Dr.Sankara Avudayappan.A.P.,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY OF SERUM URIC ACID AT PRESENTATION AS AN INDICATOR OF OUTCOME AMONG ACUTE ISCHEMIC STROKE PATIENTS" - NO. (II) 04032016.**

The following members of Ethics Committee were present in the meeting hold on **22.03.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.P.Raghumani,MS, Dept.of Surgery,RGGGH,Ch-3	: Member
5.Dr.Baby Vasumathi, Director, Inst. of O&G,Ch-8	: Member
6.Prof.M.Saraswathi,MD.,Director, Inst.of Path,MMC,Ch-3	: Member
7.Prof.Srinivasagalu,Director,Inst.of Int.Med.,MMC,Ch-3	: Member
8.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
9.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
10.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

MADRAS MEDICAL COLLEGE
31 MAR 2016
CHENNAI-600 003

Received ONE COPY from office.
Dr. (SANKARA AVUDAYAPPAN).

INFORMATION SHEET

We are conducting a study on "A STUDY OF SERUM URIC ACID AT PRESENTATION AS AN INDICATOR OF OUTCOME AMONG ACUTE ISCHEMIC STROKE PATIENTS" among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your co- operation to undergo CT Brain, Serum Uric Acid, and other relevant investigations as per needs may be valuable to us.

The purpose of the study is to understand the outcome and prognosis among acute ischemic stroke patients on the basis of their uric acid levels at presentation.

We are selecting certain cases and if you are found eligible, we would like to perform extra tests and special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator

Signature/left thumb impression of Participant

Date : 4/3/16

Place : Chennai.

ஆராய்ச்சியில் பங்கேற்பவர்கான தகவல் அறிக்கை

ஆராய்ச்சியின் தலைப்பு : இரத்தக் குழாய் அடைப்பினால் ஏற்படும் பக்கவாத நோயாளிகளின் அனுமதியின்போது உள்ள இரத்த யூரிக் அமிலத்தின் அளவுகள் – நோயின் தன்மை மற்றும் முன்னேற்றத்தில் வகிக்கும் பங்கினை பற்றிய ஆய்வு.

பங்குகொள்பவரின் பெயர் :

ஆராய்ச்சி செய்பவரின் பெயர் : சங்கர ஆவுடையப்பன், ஆ.பே.

இடம் : ராஜீவ் காந்தி அரசு பொது மருத்துவமனை
சென்னை – 600 003.

இந்த ஆராய்ச்சி / ஆய்வு / செய்முறை / சோதனையில் தாங்கள் பங்கேற்க அழைக்கிறோம். இந்த தகவல் அறிக்கையில் கூறப்பட்டிருக்கும் தகவல்கள் தாங்கள் இந்த ஆராய்ச்சியில் பங்கேற்கலாமா, வேண்டாமா என்பதை முடிவு செய்ய உதவியாக இருக்கும். இந்த படிவத்தில் உள்ள தகவல்கள் பற்றி உள்ள சந்தேகங்களை நீங்கள் தயங்காமல் கேட்கலாம்.

இந்த ஆய்வின் நோக்கம் என்ன ?

இரத்த யூரிக் அமிலத்தின் அளவுகள், இரத்த குழாய் அடைப்பினால் ஏற்படும் பக்கவாத நோயாளிகளின் நோய் தன்மை மற்றும் முன்னேற்றத்தினை கண்டறிதல்.

ஆய்வு முறைகள் :

விரிவான நோய்க் குறிப்புகளும், மருத்துவ பரிசோதனைகளும் செய்யப்படும். நோயாளிகள், அவர்கள் சம்மதத்திற்கு பின் குருதியில் உள்ள யூரிக் அமிலத்தின் அளவுகள், மூளைக்கான சி.டி. ஸ்கேன் ஆகியவற்றை பரிசோதித்து நோயாளிகளுக்கு ஏற்றவாறு தேவைப்படும் மற்ற பரிசோதனைகளை மேற்கொள்ளுதல்.

ஆய்வினால் மக்களுக்கு ஏற்படும் நன்மைகள் :

இந்த ஆய்வின் முடிவில் கிடைக்கும் தகவல்கள் சமுதாயத்திற்கு பயனுள்ளதாகவும், எதிர் காலத்தில் நோயாளிகளுக்கு மருத்துவ தீர்வாகவும் அமையும்.

தங்களிடமிருந்து பெறப்படும் தகவல்களின் நம்பிக்கை தன்மை :

தங்களிடமிருந்து பெறப்படும் தகவல்கள் பாதுகாக்கப்படுவதற்கான முழு உரிமையும் தங்களுக்கு உண்டு.

PATIENT CONSENT FORM

Study Detail : **"A STUDY OF SERUM URIC ACID AT PRESENTATION AS AN INDICATOR OF OUTCOME AMONG ACUTE ISCHEMIC STROKE PATIENTS"**

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

In Patient Number :

Patient may check (☐) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study ❑

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ❑

Signature/thumb impression
Patient's Name and Address:

Signature of Investigator
Study Investigator's Name:
Dr. SANKARA AYUCAYAPPAN.A.P.

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு: இரத்தக் குழாய் அடைப்பினால் ஏற்படும் உக்கவாத நோயாளிகளின் அனுமதியின்போது உள்ள இரத்த யூரிக் அமிலத்தின் அளவுகள் – நோயின் தன்மை மற்றும் முன்னேற்றத்தில் வகிக்கும் பங்கினை பற்றிய ஆய்வு.

ஆராய்ச்சி செய்பவரின் பெயர்: சங்கர ஆறுமுகப்பாண்டியன், ஆ.மே.

ஆராய்ச்சி மையம்: ராஜீவ் காந்தி அரசு பொது மருத்துவ மனை, சென்னை-600 003.

..... எனும் நான் எனக்கு கொடுத்துள்ள தகவல் தாளை படித்து புரிந்து கொண்டேன். நான் பதினெட்டுவயதை கடந்துள்ளதால், என்னுடைய சுய நினைவுடனும், முழு சுதந்திரத்துடனும் இந்த ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

1. நான் எனக்கு அளிக்கப்பட்ட ஒப்புதல் படிவத்தையும், தகவல்களையும் படித்து புரிந்து கொண்டேன்.
2. ஒப்புதல் படிவத்தில் உள்ள தகவல்கள் எனக்கு விளக்கி கூறப்பட்டன.
3. ஆய்வின் தன்மை பற்றி எனக்கு விளக்கப்பட்டது.
4. என்னுடைய உரிமைகளையும், பொறுப்புகளையும் ஆராய்ச்சியாளர் விளக்கிக் கூறினார்.
5. நான் இதுவரை எடுத்துள்ள / எடுத்து கொண்டிருக்கும் அனைத்து விதமான சிகிச்சை முறைகளையும் ஆராய்ச்சியாளரிடம் கூறியுள்ளேன்.
6. இந்த ஆராய்ச்சியினால் ஏற்படும் தீமைகள் பற்றி விளக்கப்பட்டன.

இடம்:

நான்: கையொப்பம்

MASTER CHART

NAME	AGE	SEX	IP NO	SHT	D M	SMOKING	DYSLIPIDEMIA	CAD	CT EVIDENCE-TERRI	URIC ACID	GLASGOW OUTCOME SCALE
JEYARAMAN	70	MALE	68740	Y	N	N	N	N	MCA	5	4
ANBARASU	55	MALE	63112	Y	N	N	N	N	MCA	11	2
PONNUSAMY	55	MALE	63127	Y	N	N	N	N	MCA	6	4
DURAIYAPPAN	80	MALE	63171	Y	N	N	N	N	MCA	10.5	3
RANGANATHAN	70	MALE	63164	Y	N	N	N	N	MCA	8.4	4
VISWANATHAN	67	MALE	63032	Y	N	N	N	N	MCA	8	3
SATHASIVAM	81	MALE	62994	Y	Y	N		N	MCA	7.8	3
PANDIYAN	50	MALE	60409	N	N	Y	N	N	PCA	9.5	3
BABU RAO	64	MALE	60177	Y	N	N	N	N	MCA	10	2
SAMBASIVAM	70	MALE	57684	Y	Y	N	N	N	PCA,MCA	4.5	4
RANGARAJAN	59	MALE	68723	Y	N	N	N	N	MCA	13	2
SHANKAR	48	MALE	68924	N	N	N	Y	N	MCA	6	5
DEVARAJ	45	MALE	68954	N	N	N	N	N	MCA	7	3
KRISHNAMOORTHY	45	MALE	68683	N	N	N	N	N	MCA	9	3
SHANMUGAM	70	MALE	66423	Y	Y	N	N	N	MCA	11	2
RAJU	55	MALE	69003	Y	N	N	Y	N	ACA	9	3
RAJAN	67	MALE	68980	Y	N	N	N	N	ACA,MCA	4.5	4
UDHAYAKUMAR	51	MALE	70463	N	Y	N	N	Y	MCA	3.8	5
THAVEETHU	47	MALE	71714	Y	N	N	N	N	ACA	6.5	4
MUNUSAMY	70	MALE	71772	Y	N	N	N	N	PCA	8	4
CHAND BASHA	48	MALE	57423	N	N	N	Y	N	MCA,ACA	9.5	4
SABAPATHY	55	MALE	91279	Y	N	N	N	N	MCA	13	2
NARAYANADOSS	80	MALE	91273	Y	Y	N	N	N	MCA	11	2
CHANDRAN	64	MALE	94010	Y	N	Y	N	Y	MCA	6	3
ETHIRAJ	55	MALE	94041	Y	Y	N	N	N	MCA	5.5	4
THAMIM BASHA	40	MALE	96424	N	N	Y	N	N	PCA,MCA	4.5	4
SRINIVASAN	66	MALE	96565	Y	N	N	N	N	ACA	6	4
UDHAYAKUMAR	70	MALE	96656	Y	N	N	N	N	MCA	7	4
KANNAN	27	MALE	96655	N	N	Y	N	N	MCA,PCA	8.5	3
EZHIL	45	MALE	96601	Y	N	N	N	N	MCA	13	1
RAJENDRAN	46	MALE	82910	Y	N	N	N	N	MCA	10	2
RANGAIAH	52	MALE	83015	Y	N	N	N	N	MCA	8	3
ROSEDEVAN	65	MALE	83033	Y	N	N	N	Y	MCA	9	3
MAGESH	47	MALE	96591	Y	N	N	N	N	MCA	8.5	4

RAJASEKAR	52	MALE	85701	Y	Y	N	N	N	ACA	7	4
SARAVANAN	60	MALE	85693	Y	Y	N	N	N	PCA	8.5	3
DHANAPANDIYAN	47	MALE	86491	Y	N	N	N	Y	MCA	13	1
RAMALINGAM	80	MALE	88473	Y	Y	N	N	N	MCA,PCA	9	3
SANKAR	75	MALE	88511	Y	Y	Y	Y	N	MCA	6	4
JAYARAMAIAH	65	MALE	88549	Y	N	N	Y	N	MCA	9	2
NANDHAKUMAR	38	MALE	74578	Y	Y	N	N	Y	MCA	11	2
PERUMAL	84	MALE	74551	N	Y	N	N	N	MCA	14	1
VANDHANAM	65	MALE	77426	N	N	N	N	N	ACA	8	3
BAKTHAVATSALAM	67	MALE	77354	N	N	N	N	Y	MCA	7	4
SEKAR	60	MALE	77411	Y	N	N	N	N	MCA	13	2
PALANI	56	MALE	79980	N	N	Y	N	N	ACA	4.8	5
MUNUSAMY	65	MALE	80169	N	N	N	N	N	MCA	9.5	3
SAKTHIVEL	48	MALE	82934	N	N	Y	N	N	MCA	9	3
CHINNATHAMBI	62	MALE	82820	Y	N	N	N	N	ACA	8	4
BASHA	62	MALE	83001	N	N	N	N	N	MCA,ACA	12	2
NAGAMMA	45	FEMALE	35138	Y	N	N	N	N	MCA,PCA	8	3
ANJALI	58	FEMALE	34790	N	N	N	N	N	MCA	7.5	4
PARVATHY	72	FEMALE	37982	Y	Y	N	Y	Y	MCA,PCA	7	3
SELVI	44	FEMALE	38043	N	Y	N	N	N	MCA	9.5	3
LALITHA	65	FEMALE	40721	Y	N	N	N	N	MCA	3.5	5
CHINNAMMAL	60	FEMALE	40648	Y	N	N	Y	N	PCA	11	2
AROKIASAMY	34	MALE	74452	N	N	N	N	N	PCA	8	3
BABU	63	MALE	74503	Y	N	N	N	N	ACA	8.5	3
ARJUNAN	59	MALE	74489	Y	N	N	N	N	MCA	9	2
SIVANATHAN	48	MALE	74484	Y	N	N	N	N	MCA,PCA	8.5	3

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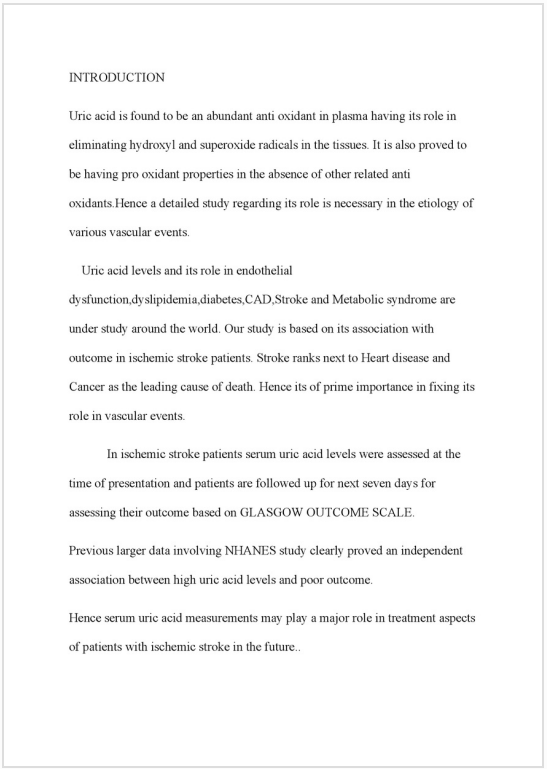


INTRODUCTION

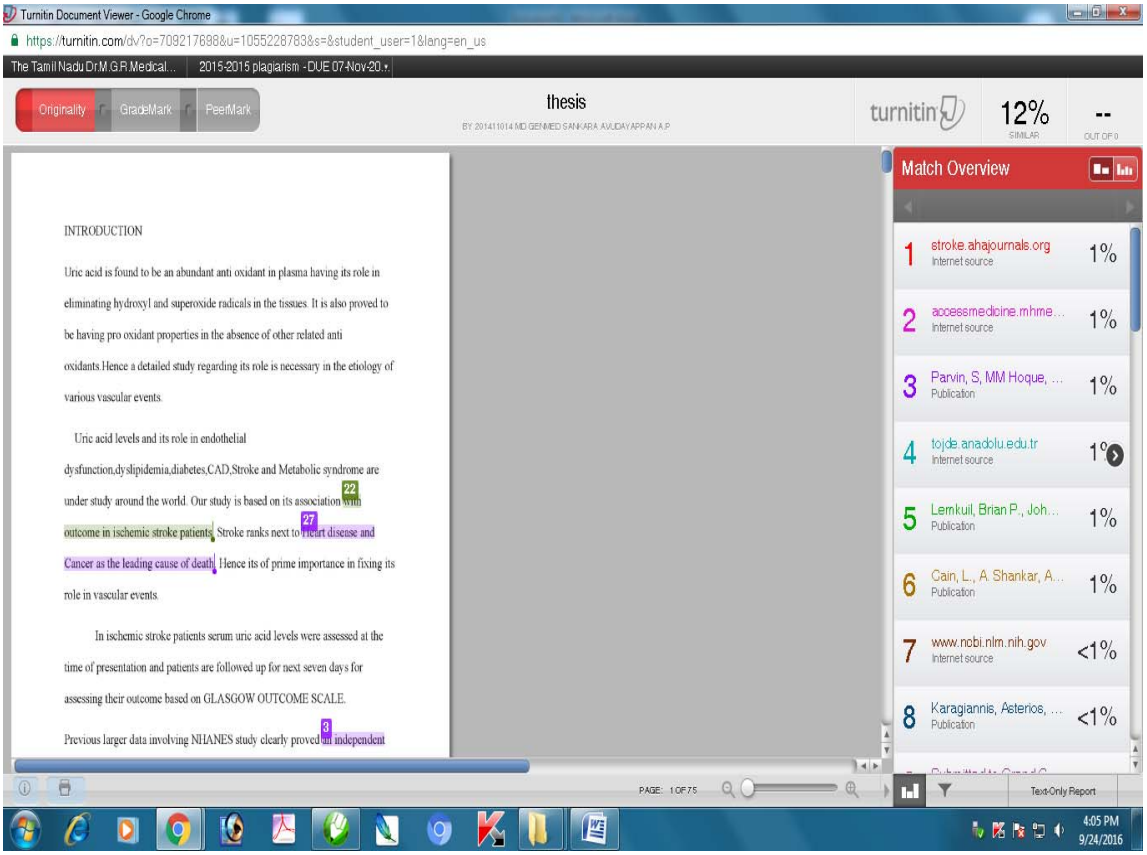
Uric acid is found to be an abundant anti oxidant in plasma having its role in eliminating hydroxyl and superoxide radicals in the tissues. It is also proved to be having pro oxidant properties in the absence of other related anti oxidants.Hence a detailed study regarding its role is necessary in the etiology of various vascular events.

Uric acid levels and its role in endothelial dysfunction,dyslipidemia,diabetes,CAD,Stroke and Metabolic syndrome are under study around the world. Our study is based on its association with outcome in ischemic stroke patients. Stroke ranks next to Heart disease and Cancer as the leading cause of death. Hence its of prime importance in fixing its role in vascular events.

In ischemic stroke patients serum uric acid levels were assessed at the time of presentation and patients are followed up for next seven days for assessing their outcome based on GLASGOW OUTCOME SCALE.

Previous larger data involving NHANES study clearly proved an independent association between high uric acid levels and poor outcome.

Hence serum uric acid measurements may play a major role in treatment aspects of patients with ischemic stroke in the future..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